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73

# INDEX TO VOLUME XX.

NEW SERIES.

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#### ORIGINAL ARTICLES.

Acute Poisoning by Arsenic, with Subsequent Multiple Neuritis (On a Case of). By Philip Meierowitz, M.D.	178
Amyotrophic Lateral Sclerosis, with Bulbar Paralysis and Degeneration in Goll's Columns. A Contribution to the Pathology of the Primary Combined System Diseases. By Ludvig Hektoen, M.D.	145
Angular Gyrus with Left-sided Ptosis (Note on a Case of Softening of the Right). By C. A. Herter, M.D.	18
Arsenic, with Subsequent Multiple Neuritis (On a Case of Acute Poisoning by). By Philip Meierowitz, M.D.	178
Ataxia; A Critical Examination of the Evidence, with Reports of New Alleged Cases (Traumatism as a Cause of Locomotor). By Morton Prince, M.D.	77
Beri-Beri (Report of Three Cases of). By F. X. Dercum, M.D.	103
Cells (Evolution of the Nerve). By Ramon y Cajal.	775
Cerebral Cortex to Sensation (Relations of the). By W. H. Thomson, M.D., L.L.D.	333
Cerebral Tumors Mistaken for Paresis, with Autopsy (Report of Two Cases of). By L. Pierce Clarke, M.D.	273
Circular Insanity; Report of Three Cases. By W. F. Drewry, M.D.	223
Differential Diagnosis Between Localized Neuritis, Rheumatism and Some Other Conditions That Give Similar Symptoms. By Louis Faugères Bishop, M.D.	401
Digitus Mortui for Four and a Half Years' Duration (A Case of). By C. E. Stanley, M.D.	20
Duplex Personality; Its Relation to Hypnotism and to Lucidity. By R. Osgood Mason, M.D.	420
Epilepsy; Seven Years After. Complete Recovery (Further Report of Operation for). By J. Madison Taylor, M.D.	231
Evolution of the Nerve Cells. By Ramon y Cajal.	774
Exhibiting Symptoms of Facial Hemiatrophy and Jacksonian Sensory Epilepsy (A Case of). By Theodore Diller, M.D.	284

Facial Hemiatrophy and Jacksonian Sensory Epilepsy (A Case of Exhibiting Symptoms of). By Theodore Diller, M.D.	284
General Paresis, with Special Reference to the Study of the Relations Between the Temperature, Pulse-rate, and Respiration (Note in a Case of). By Ralph Wait Parsons, M.D.	100
Gold in Two Cases of Nervous Diseases (Successful Use of). By J. J. Alexander Wade, M.D.	330
Gyrus with Left-sided Ptosis (Note on a Case of Softening of the Right Angular). By C. A. Herter, M.D.	18
Hemiatrophy and Jacksonian Sensory Epilepsy (A Case Exhibiting Symptoms of Facial). By Theodore Diller, M.D.	284
Histological Conformation of the Medulla (The). By Wm. C. Krauss, M.D., F.R.M.S.	12
Idiocy (The Influence of Heredity on). By Martin W. Barr, M.D.	341
Influence of Heredity on Idiocy (The). By Martin W. Barr, M.D.	341
Lepra Anæsthetica and Syringomyelia in Colombia. By Albert S. Ashmead, M.D.	770
Localized Neuritis, Rheumatism and Some Other Conditions That Give Similar Symptoms (Differential Diagnosis Between). By Louis Faugères Bishop, M.D.	401
Locomotor Ataxia; A Critical Examination of the Evidence with Reports of Three New Alleged Cases (Traumatism as a Cause of). By Morton Prince, M.D.	77
Locomotor Ataxia—Sudden Onset and Unusual Symptoms. Posterior Sclerosis of Similar Symptomatology in Patient's Wife. By F. Savary Pearce, M.D.	8
Medulla (The Histological Conformation of the). By Wm. C. Krauss, M.D., F.R.M.S.	12
Melancholia Attonita (Report of a Case of). By Thomas E. Bamford, M.D.	357
Meryasmus or Rumination; with a Report of Two Cases. By David Reisman, M.D.	359
Morbid Hereditary and Predisposition to Insanity of the Man of Genius (On the). Warren L. Babcock, M.D.	749
Myxedema and Exophthalmic Goitre in Sisters; with Remarks on the Symptomatology of the Latter Disease. By A. R. Oppenheimer, M.D.	213
Naming Centre; with Report of a Case Indicating Its Location in the Temporal Lobe (The). By Chas. K. Mills, M.D.	1
Nerve Cells (Evolution of the). By Ramon y Cajal	775
Neuritis, Rheumatism and Some Other Conditions That Give Similar Symptoms (Differential Diagnosis Between Localized). By Louis Faugères Bishop, M.D.	401
Neurological Bust (A). By Wm. C. Krauss, M.D.	101
Operation for Epilepsy; Seven Years After Complete Recovery (Further Report of). By J. Madison Taylor, M.D.	231
Problem in Neurology. Peculiar Iris Reaction, with Post Neuritic Optic Atrophy (A). By George M. Gould, M.D.	258
Progressive Muscular Atrophy. Most Marked in the Rhomboid and Shoulder Muscles, but Also Involving the Muscles of the Tongue and Face, and External Ocular Muscles. By J. Torrance Rugh, M.D., and Chas. K. Mills, M.D.	354
Pseudo-Muscular Hypertrophy (A Case of). By Augustus A. Eschner, M.D.	28
Right Angular Gyrus with Left-sided Ptosis (Note on a Case of Softening of the). By C. A. Herter, M.D.	18
Sclerosis, with Bulbar Paralysis and Degeneration in Goll's Columns. A Contribution to the Pathology of the Primary Combined System Diseases (Amyotrophic Lateral). By Ludvig Hektoen, M.D.	143

Softening of the Right Angular Gyrus with Left-sided Ptosis (Note on a Case of). By C. A. Herter, M.D.....	18
Tabes Associated with Post Hemiplegic Athetosis and Unilateral Reflex Iridoplegia (A Case of). By Joseph Collins, M.D.....	294
Tabes (Valvular Disease of the Heart in). By Pearce Bailey, M.D.....	290
Tetanus (Note on a Case of). By Philip Meirowitz, M.D.....	235
Traumatism as a Cause of Locomotor Ataxia; A Critical Examination of the Evidence, with Reports of Three New Alleged Cases. By Morton Prince, M.D.....	77
Valvular Disease of the Heart in Tabes. By Pearce Bailey, M.D....	290
Waiter's Paralysis (A Case of). By Edward C. Runge, M.D.....	241

### *SOCIETY REPORTS.*

#### NEW YORK NEUROLOGICAL SOCIETY.

Meeting of November 26, 1894: Presentation of Cases—Notes on a Case of Acute Poliomyelitis in a Hen—Exhibition of Microphotographs of Nerve Cells (Golgi's Stains)—Some of the Important Aspects of the Therapeutics of the Nervous System....	50
Meeting of December 4, 1894: Tumor of the Brain—Two Cases of Cerebral Syphiloma—A Case of Muscular Dystrophy.....	130
Meeting of January 8, 1895: A Case for Diagnosis—A Case of Acute Arsenical Poisoning, with Subsequent Multiple Neuritis—Three Cases of Unilateral Lesion of the Spinal Cord—Tumor of the Optic Thalamus, with Autopsy—Tumor of the Aqueduct of Sylvius, with Presentation of Specimen.....	202
Meeting of February 5, 1895: Charcot Monument Fund—Athetosis with Beginning Tabes—A Contribution to the Subject of Tumors of the Spinal Cord, with Remarks on their Diagnosis and Their Surgical Treatment, with a Report of Six Cases, in Three of Which the Tumor was Removed..	258
Meeting of March 5, 1895: Charcot Monument Fund—A Case of Tabes Associated with Post-Hemiplegic Athetosis and Unilateral Reflex Iridoplegia—Palmus.....	317
Meeting of April 2, 1895: Tumor of the Brain; Successful Operation—Relations of the Cerebral Cortex to Sensation .....	385
Meeting of May 7, 1895: An Unusual Case of Lead Poisoning—An Intracranial Gumma—Report of a Case of Lead Paralysis, with Special Reference to Cytological Changes in the Nervous System and the Distribution of the Lead.....	441
Meeting of October 1st, 1895: Neuritis of the Brachial Plexus as a Sequel of Pneumonia—Atypical Hereditary Cerebellar Ataxia—A Cystic Tumor—Amyotrophic Lateral Sclerosis—A Report of a Series of Cases of Multiple Neuritis in Infants in the city of Bridgeport, Conn. ....	787

#### PHILADELPHIA NEUROLOGICAL SOCIETY.

Meeting of November 26, 1895: Exhibition of Anatomically Correct Plaster Casts of Brain of Ourang-Outang and Brain of Chimpanzee—Exhibition of the Brains of the Chimpanzee and Ourang-Outang Belonging to Prof. H. C. Chapman—Specimens from Cases of Pernicious Anemia, Showing Lesions of the Spinal Cord—Lesion of the Naming Centre, with Report of a Case Indicating Its Location in the Temporal Lobe—Locomotor Ataxia with Sudden Onset, and Unusual Initial Symptoms. Posterior Sclerosis of Similar Symptomatology in Patient's Wife—Exhibition of Brain Tumors.....	58
Meeting of December 17, 1895: Three Cases of Beri Beri—Exhibition of Patient .....	136
Meeting of January 28, 1895: The Alleged Reflex Causes of Nervous Diseases. ....	266



Meeting of February 25, 1895 : Neuritis During and After Typhoid Fever—Some Cases of Sexual Perverts .....	327
Meeting of March 25, 1895 : A Case of Jacksonian Sensory Epilepsy—A Case of Pseudo-Muscular Hypertrophy—Meryasmus or Rumination—A Case of Uniform Arrest of Development of the Lower Extremity Without any Accompanying Abnormal Conditions—A Case of Progressive Muscular Atrophy, Showing Especially Atrophic Paralysis of the Rhomboid and Supinate Muscles, the Tongue and the Face—A Brain, Showing an Old Cyst, Involving Chiefly the Third Frontal and the Lower Extremity of the Central Convolutions—Huntington's Chorea—Nerve Suture.....	391
TWENTY-FIRST ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION.	
Address by the President. By Philip Coombs Knapp, A.M., M.D.....	457
Hysterical Amblyopia and Amaurosis. Report of Five Cases Treated by Hypnotism. By J. Arthur Booth, M.D .....	464
A Case of Complete Hysterical Anesthesia in the Male. By George J. Preston, M.D.....	475
Two Cases of "Railway Spine," with Autopsy. By F. N. Dercum, M.D.....	481
A Lantern Exhibition of Photo-Micrographs of Nervous Histology; Golgi Stains. By M. Allen Starr, M.D.....	497
A Lantern Exhibition of the Medulla Oblongata of a Chimpanzee, with Other Specimens. By Edward Wyllys Taylor, M.D.....	497
Exhibition of the Brain of a Chimpanzee. By Thomas Dwight, M.D. ....	497
The Part of Inhibition in the Physiology of Respiration. By William Townsend Porter, M.D .....	497
Hyperostosis Cranii (Ophthalmogaly), with Illustrations. By James J. Putnam, M.D. ....	500
A Cast of a Brain and a Brain Preserved in Alcohol. By F. T. Miles, M.D.....	506
Cortical Localization in the Light of Recent Researches into the Minute Anatomy of the Cortex. By Charles K. Mills, M.D. ....	527
Report of a Case of Tumor of the Cerebellum, with Autopsy. By E. D. Fisher, M.D., and John F. Erdmann, M.D.....	544
Three Cases of Tumor of the Brain, with Autopsy. By George J. Preston, M.D.....	552
Presentation of a Brain Tumor. By George L. Walton, M.D....	553
A Case of Injury to the Left Angular Gyrus. By C. Eugene Riggs, M.D. ....	561
The Pathology of Hereditary Chorea. Report of a Case, with Autopsy. Record of Anomalies in a Degenerate Brain. By Charles L. Dana, M.D.....	565
The Biological and Morphological Constitution of Ganglionic Cells, as Influenced by Section of the Spinal Nerve Roots or Spinal Nerves. To Which is Appended Some Remarks on Localization. By B. Onuf (Onufrowicz), M.D.....	597
Insanity and Phthisis; Their Transmutation, Concurrence and Co-existence. By H. A. Tomlinson, M.D. ....	613
The Diagnosis of Pachymeningitis Interna Hemorrhagica. By William N. Bullard, M.D.....	681
A Case of Incipient Locomotor Ataxia and Monoplegia from Focal Lesion of the Internal Capsule in the Same Patient. By Morton Prince, M.D. ....	685
A Case of Landry's Paralysis, with Autopsy. By Morton Prince, M.D.....	686
A Contribution to the Pathology and Morbid Anatomy of Amvo	

trophic Lateral Sclerosis, with a Report of Two Cases, with Autopsy, in One of Which, Observed by Dr. C. L. Dana, a Terminal Tubercular Cervical Myelitis and Perforating Necrosis of the Dorsal Cord Was Found. By Joseph Collins, M. D. ....	688
Report of a Case of Peroneal Muscular Atrophy, with Autopsy. By Wm. C. Krauss, M.D. ....	693
Two Cases of Writer's Cramp. By James W. Putnam, M.D. ....	696
The Etiology of Obstetrical Paralysis. By G. L. Walton, M.D. ....	702
Description of Baths, with Exhibition of Photographs and Drawings. By Ralph L. Parsons, M.D. ....	704
The Cerebral Fissures of Two Philosophers, Chauncey Wright and James Edward Oliver. By Burt G. Wilder, M.D. ....	706
Irrigation of the Posterior Cerebral Fossa for the Relief of Basilar Meningitis. By J. T. Eskridge, M.D. ....	708
The Home Treatment of Insanity. By H. M. Bannister, M.D. ....	718
The Association of Tabes and Paralytic Dementia. Report of Five Cases. By Theodore Diller, M.D. ....	729
A Study of Three Cases of Tumor of the Brain, in Which Operation of the Brain was Performed—One Recovery, Two Deaths. By Leo Stieglitz, M.D. ....	730

### ASYLUM NOTES.

Among the Hospital Reports, 370—A Study of the Degenerative and Destructive Disease of the Lungs Among the Insane, 424—A Study of the Hospital Reports, 424—A Synopsis of One Year's Admissions, 797—Asylum Notes, 508—A New Journal That Ought to Be Good, 805—Comments on Hospital Reports, Notes, etc., 181—Dr. Daniel Clarke, 735—Dr. Richard Dewey, 735—Hospitals for Acute Cases Only, 662—Investigation in New York, 662—New Appointments, 735—Notes, 113—Points of View in Clinical Study of Psychiatric Work, 181—Pathological Supplement to the Annual Report of the Government Hospital for the Insane, 1893, 801—Sketch of the Rochester State Hospital for the Insane, 243—Some Reasons Why More Original Investigations and More Literary Work is Not Done by the Physicians in Large Public Hospitals for the Insane, with a suggestion of Certain Remedial Measures, 22—Synopsis of One Year's Admissions, 781—The Boundary Lines of Insanity, 735—The Expiscation of Acute Delirium, 424—The Medico-Legal Society of New York, 735—The New York Lunacy Commission's Report, 113—The Present Standing of Training Schools for Nurses in Hospitals for the Insane, 584—Trained Nurses to Travel with Patients, 662—Training Schools, 113—Transfer of New York City Insane, 662.
--

### BOOK REVIEWS.

Anatomie des Centres Nerveux. By J. Dejerine. ....	140
Anniversary of the Standard Dictionary. ....	830
Appendix to Dundlison's Medical Dictionary. ....	830
A Text-Book on Nervous Diseases by American Authors. Edited by F. N. Dercum, A.M., M.D. ....	815
Degeneration. By Max Nordau. ....	453
Diagnostic Neurology. By A. B. Shaw, M.D. ....	212
Die Leitungsbahnen im Gehirn und Rückenmark. Von W. von Bechterew. ....	69
Diet in Disease. By W. Gilman Thompson, M.D. ....	827
Leçons sur les Maladies Nerveuses. By E. Brissand. ....	521
Lecture on Appendicitis and Notes on Other Subjects. By Robert T. Morris, A.M., M.D. ....	828

Injuries and Diseases of the Nerves. By John K. Mitchell, M.D.	524
Myxœdema and the Thyroid Gland By John D. Gimlette, M.R.C.S., L.R.C.P.	529
Nervous Diseases of Children. By B. Sachs, M.D.	455
Neurological Contributions. By C. J. Möbius, M.D.	675
On the Relation of Diseases of the Spinal Cord to the Distribution and Lesion of the Spinal Blood Vessels. By R. T. Williamson, M.D., M.R.C.P., etc.	524
Practical Dietetics. With Special Reference to Diet in Disease. By W. Gilman Thompson, M.D.	527
Practical Urinalysis and Urinary Diagnosis By Charles W. W. Purdy, M.D.	210
Relations of Diseases of the Eye to General Diseases. By Max Kuies.	451
The Art of Massage. By J. H. Kellogg.	679
The Eye and its Relation to Health. By Chalmer Prentice.	525
Thinking, Feeling, Doing. By E. W. Scripture, Ph.D.	825
Uric Acid as a Factor in the Causation of Disease. By Alexander Hay, M.A., M.D.	141

### PERISCOPE.

Anæsthesia Produced During Sleep (Chloroform), 742—Acromegaly, 743—Aeteris at the Base of Brain in Sane and Insane (Changes on the 31—Alexia with Right-sided Homonymous Hemianopsia (A Case of), 39—Achilles Tendon Reflex (The Diagnostic Importance of), 40—Akinesia Algera, 121—Akinesia Algera, 122—Amusia: Musical Aphasia, 123—Atasia Abasia and Its Treatment, 123—Apoplexy in its Relation to the Temperature of the Body with a Consideration of the Question of Heat Centres, 195—"Anxiety Neurosis" (The), 197—Acromegaly (A Case of), 198—Anthropological Significance in the Negro Brain (A Finding of), 250—Alcohol on Sexual Perversions, Epilepsy and Other Psychological Anomalies (Influence of), 314—Apoplexy (Observations on), 383—Appearance of Secondary Degeneration in the Single Tracts of the Cord On the Time of), 591—Acute Mania, 592—Acute Non-Purulent Encephalitis (The Prognosis of), 594—Aetiology of Dementia Paralytica with Especial Consideration of Syphilis (Contribution to the), 673—Amaurosis by the Extract Filicis Maris (Note on the Production of), 810—Abortive Acromegaly with Macroglossia, 812—Anorexia Hysterica, 814.
Baker, Dr. W. H. Baker, 383—Bulbar Manifestations in Siringomyelia, 438.
Co-ordination (On a Symptom Complex of Congenital Apparently Acquired, Disturbance of), 741—Chloroform Anæsthesia Produced During Sleep, 742—Craniectomy (Therapeutic Effect of an Exploratory), 745—Cerebro-Spinal Meningitis (The Pathological Anatomy of), 31—Chorea Chronica Progressiva Hereditaria (Pathological Anatomy of), 32—Caries of the Spine, Causing Compression of the Cord Relieved by Laminectomy, 36—Circular Neurasthenia, Central Localizations (Some Disputed Points Regarding the Doctrine of), 189—Causation of Hemiatrophia Facilis Progressiva (The), Cortex Cerebri (A New Method of Dividing the Surface of the), 247—Cerebral Optical Centres, 248—Cerebral Sclerosis Following Influenza, 313—Cerebral Tumor Without Headache and with Negative Ophthalmoscopic Examination (Large), 315—Cerebral Anorexia, 383—Cerebellar Heredo-Ataxia, 436—Cerebral Hemorrhage as a Sequel of Enterocolitis, 437—Congress of German Surgeons, 513—Cerebral Palsies of Childhood, 807.
Degeneration and Its Stigmata, 31—Duboisine in Morphinism, 37—Duboisine in Mental Medicine (Subcutaneous Injections of Sul-

- phate of), 37—Differential Diagnosis Between Injury of the Cauda Equina and the Lumbar Enlargement, 41—Dyspepsia (Nervous), 198—Differential Diagnosis of Traumatic Intracranial Lesions (The), 376—Disturbances of Sensibility of the Globe of the Eye and Its Appendages in Locomotor Ataxia (The), 434—Doctrine of the Infectious Origin of Acute Delirium (Contribution to the), 518—Descending Cerebellar Tracts, 668—Degenerations Following Cerebellar Lesions (On the), Preliminary Communication, 672—Diffuse Sclerosis of the Brain and Spinal Cord, 811.
- Encephalasthenia (The Differential Diagnosis of), 34—Extra-Medullary Spinal Cord Tumor (The Diagnosis of), 37—Electric Brush (The), 38—Epilepsy (Treatment of), 257—Epilepsy, with Special Reference to the Use of Opium (Treatment of), 309—Epilepsy Cured by Curare, 311—Enteritis (Nervous Complications Caused by Mucous Membranous), 314—Experimental Study of the Nervous Manifestations of Chronic Arsenical Poisoning (Contribution to the), 438—Exhibition of Three Microcephalic Children, 438—Electricity in Medicine, &c.
- Friedrich's Disease with Necropsy (A Case of), 36—Facial Paralysis (Rapid Cure of), 41—Fatigue of the Human Muscles Caused by the Action of the Nerve Poisons (Experimental Researches on the), 249—Fascicle and the Cerebral Cortex (Reil's), 590—Fever of Nervous Origin, 594—Fine Anatomy of the Optic Roof of the Teleostei Fish (On the), 670—Facial Hemiatrophy, 671—Further Researches on the Ulnar Symptoms in Lunatics, 807.
- Galvanization of the Brain, 119—Ganglionic Cells (Restitution of Pathologically Changed), 120—Grave's Disease (Etiology of), 124—Gangrene of the Skin of Hysterical Origin (Disseminated and Successive), 127—Glioma of the Cerebral Ventricles (A Case of Extensive Ependymal), 192—Glioma in the Dorsal Portion of the Pons (A Case of), 193—Guayacol (On the Analgesic Properties of), 201—Glio Sarcoma of the Cerebellum, 256—Gold as a Therapeutic Agent, 257—Gerlier's Vertigo (A Case of), 382.
- Histological Alterations in Cerebral Cortex in Some Mental Disease, 32—Hæmatoma Auris, 198—Hot Baths in Protracted Cerebro-Spinal Meningitis, 200—Hypnotics (Notes on), 257—Hysterical Nystagmus (Is there), 306—Hæmi-anæsthesia and Hemiparaplegia from Medullary Concussion (Crossed), 376—Hereditary Ataxia, 381—Hysteria of Gastro-Intestinal Origin, 674.
- Insomnia in Surgery, 745—Isolated Paræsthesia in the Distribution of the Nervous Cutaneous Femoral Externus, 313—Intracranial Hæmatoma with Collateral Paralysis, 515—Investigation of the Changes Found in the Ganglionic Cells in Acute Alcoholic Poisoning (Experimental), 520—Influenzal Meningitis, 595—Incipient Meningitis (A Constant Sign in), 595—Infantile Giantism with Tumor of the Testicle (A Case of), 673.
- Kahlbaum's So-called Katatonía (A Case of), 814.
- Lesion of the Tonsil, Velum Palati and Uvula, Complicating Multiple Neuritis (A Case of Neuropathic), 124—Laryngeal Paralysis in Chronic Nervous Disease, 126—Lesion of Tabes (The Primitive), 194—Lesions of the Spinal Cord (The Reflexes on Total Transverse), 194—Lesions of the Crura Cerebri in Tabes (On the), 252—Lead Poisoning with Extremely Rare Symptoms (A Case of), 575.
- Myxœdema (The Treatment of), 37—Mutual Relation Between Anæsthesia and Sensory Anæsthesia, (The), 118—Muscular Pathology (Contributions to), 119—Multiple Neuritis (A Case of), 125—Myxœdema (Examination of the Blood in), 308—Myasthenia Gravis Pseudo-Paralytica, 312—Migraine (The Equivalent of), 312.
- Nervous Diseases (On Little Known Special Manifestations), 739—Neuritis and Tabes (A New Theory of the Causes of Some Nervous

- Diseases, Especially of), 33—Neuro-Psychoses (The Warding Off), 129—Nomenclature in the Anatomy of the Nerve Cells, 260—Neurons and their Connection Simply by Contact (The Theory of), 589—Neurasthenia Insomnia (The Treatment of), 509—Neuritis Caused by Vascular Diseases (On a Form of), 813.
- Optic Neuritis as a Sign of Brain Tumor, 127.
- Pathological Anatomy of Chronic Chorea (The), 592—Posterior Corpora Bigemina as Centres of Hearing, of the Voice, and of Special Movements (The), 740—Pathology of Progressive Pseudo-Hypertrophy and Muscular Dystrophy (Contributions to), 120—Paralytic Dementia, 120—Puerperal Polyneuritis, 125—Peripheral Facial Paralysis (On the Etiology of), 125—Psychosis (The Polyneuritis), 127—Pseudo-Hypertrophic Paralysis in Late Life, 196—Pathogenesis of Acromegaly, 254—Physiology of the Cerebellum, According to Fenier (Recent Studies on the), 305—Pathological Anatomical Findings on the Clivus in Insane Patients, 308—Paralysis of the Cranial Nerves Caused by Syphilis (Multiple), 314—Puerperal Neuritis, 438—Pseudo Tabes Mercurealis, 439—Periodic Paranoia (A Case of), 517—Posterior Longitudinal Fascicle (The), 590—Physiological Action of Massage, 591—Pathology and Therapy of Progressive Bulbar Paralysis, 808.
- Rule of the Facial Nerve in Secretion of Tears (On the), 192—Raynaud's Disease (An Hysterical Form of), 196—Respiratory Paths in the Cord (Crossing of the), 307—Relation of Infectious Processes to Mental Disease (The), 316—Role Played by Fixed Ideas in the Pathogenesis of Hysterical Polyuria (The), 316—Raynaud's Disease (Two Cases), 440—Researches on the Connections Existing Between the Nuclei of the Motor Nerves of the Eye on One Side, and the Posterior Longitudinal Fascicle and Lenticular Formation on the Other Side, 806.
- Syphilis of the Nervous System (Contribution to the Study of), 20—Sclerosis (Pathological Anatomy of Multiple), 30—Syringomyelia Associated with Hysteria (A Case of), 35—Spastic Spinal Paralysis as a Partial Manifestation of an Hereditary Syphilitic Affection of the Central Nervous System (The Symptom Complex of So-called), 38—Syringomyelia with Crico Arytenoidieus Posticus Paralysis and Atrophy of the Trapezius (A Case of), 126—Subcortical Tumor Treated by Operation (A Case of), 195—Syphilis Stimulating Amyotrophic Lateral Sclerosis, 199—Spasmodic Torticollis (The Operative Treatment of), 201—Scleroderma in Plagues, 308—Subacute Nuclear Ophthalmoplegia and Paralysis of the Extremities with Post-Mortem Findings (A Case of), 431—Study of Spinal Disease Occurring in the Course of Pernicious Anæmia (Further Contributions to the), 432—Study of Combined Systemic Diseases of the Cord (Anatomical Contribution to the), 434—Staining of the Nervous System (The), 519—Sulfonal in Insanity, 596—Sensible Touch (The), Temperature and Pain, and Sensory Optic, Acoustic, Olfactory, etc., Conducting Pathways and Centres, 669—Section of Both Pyramids in Dogs: Preliminary Communication, 670.
- Trinin in the Treatment of Sciatica, 743—Tea and Its Effects, 744—Tetanus Cured by the Antitoxin of Tizzoni Cattine (A Case of), 745—Tubercular Meningitis (Treatment of), 201—Traumatic Tabes and the Pathogenesis of Tabes in General (On), 254—Trophic Properties of Nerves (The), 307—Thyroid on the Amount Oxyhemoglobin in the Blood (The Influence of the), 308—Tetanus Truncatus Treated with Antitoxin, 311—Trismus and Tetanus Monotorum Treated with Tizzonis Antitoxin (Four Cases of), 311—Tetanus Treated Subcutaneous Injections of Corrosive Sublimate (A Severe Case of), 312—Traumatic Atrophy of the Serratus Magnus, 315—Trional as an Hypnotic, 379—Tabes Incipiens (A Case of), 304—

- Tabes Arrested by Blindness, 516—Trional Poisoning, 596—Thalamic Heat Centres, 806—Tlalalgie, 808—The Employment of Sparks of Opened Induction Coils for Diagnostic and Therapeutic Purposes, 808—Tobacco Mania Treatment by Hypnotism and Suggestion, 813.  
 Urine in General Paralysis (The), 199—Unilateral Disturbance of Respiration in Cerebral Paralysis, 384.  
 Voluntary Motion as Influenced by Sensory Disturbances, 375—Vasomotor Disturbance of Cerebral Origin (Half-Sided), 440—Vertigo, 592.  
 Wasting of the Facial Muscles (A Case of Unilateral, Probably Congenital), 40—Weigert-Pal Staining Method for the Central Nervous System which has been Hardened by Formalin (The Employment of), 192.

*CORRESPONDENCE.*

- Letter from E. P. Hurd, M.D. .... 526  
 Letter from R. Osgood Mason, M.D. .... 748

*MISCELLANY.*

- A Correction ..... 831  
 Annual Announcement ..... 831







THE  
**Journal**  
OF  
Nervous and Mental Disease.

**Original Articles.**

THE NAMING CENTRE, WITH THE REPORT  
OF A CASE INDICATING ITS LOCATION IN  
THE TEMPORAL LOBE.<sup>1</sup>

By CHARLES K. MILLS, M.D.,

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AND

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THE setting apart of a special area with the designation, *naming centre*, is in accordance with the views of Broadbent, Kussmaul and Charcot. According to Broadbent, the formation of an idea of any external object is the combination of the evidence respecting it received through all the senses. For the employment of this idea in intellectual operations it must be associated with and symbolized by name. The structural arrangement connected with this process, he supposes to consist in the convergence from all the receptive centres of tracts which go to a convolutional area on the sensory side of the nervous system, which may be called the *naming centre*. Its correlative motor centre is the propositionizing centre, in which names or nouns are set in framework for outward ex-

<sup>1</sup> Read at a meeting of the Philadelphia Neurological Society, November 26, 1894.

pression, and in which a proposition is realized in consciousness or mentally rehearsed. The destruction of this centre would cause loss of memory of names or nouns. As a provisional guess Broadbent placed this centre in an unnamed lobule situated on the under surface of the temporal lobe, near its junction with the occipital lobe, where he believed fibres from all the perceptive centres converge to, and end in the cortex of this region. A careful study of the entire subject of speech disturbances, including an analysis of cases already reported, will be convincing as to the necessity of a higher area for thought and speech, intermediate between the sensory or receptive centres and the motor or emissive.

Other names which have been applied to this centre or area are *idea centre* and *concept centre*. Some authorities, as Ross<sup>2</sup> and Bastian, do not consider that it is necessary to have a particular region of the brain in which concepts are elaborated and symbolized by name; but even Ross and Bastian acknowledge a special development of the cortex for concepts and names, but would not restrict it to an isolated area.

Recently a case which seems to be convincing as to the existence and localization of this naming or idea centre has fallen under our observation, one which would seem not only to support the separate localization of such a region but also to confirm Broadbent's speculation, made long ago, as to the position of this centre in the temporal lobe.

This patient was seen in consultation with Dr. Wilson Bowers. The following is a condensed history of the case and an account of the autopsy:

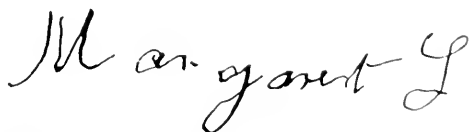
M. R., white, married, aged about forty, five years before her death complained for the first time of numbness of the back of the neck and vertigo, and had an attack of excessive vomiting, lasting two days. Nothing occurred subsequently to this, or nothing could be learned of anything occurring again until three years later, when it was noticed that she did things differently from her usual custom. Dressmaking, at which she had been successful, was poorly done; she hung upside down a certificate of membership in a society to which she belonged without realizing her error. Her appearance also changed from that of a woman in her prime to one fast advancing in years. When she arose

<sup>2</sup> Ross: Aphasia, in Wood's Med. and Surg. Monographs, Vol. vi., No. 1, April, 1890.

in the morning of December 3, 1893, she acted very strangely, was very nervous, imagined that she saw a light, could not read, and remarked that "she felt like killing her daughter." In the evening of the day she was seized with convulsion; frothed at the mouth and was unconscious. The next day she was very forgetful, but without apparent speech trouble. She was confined to bed for two weeks, during which time she aged very rapidly, and commenced to complain of severe headache and of numbness in the neck. Soon after this verbal amnesia became very evident.

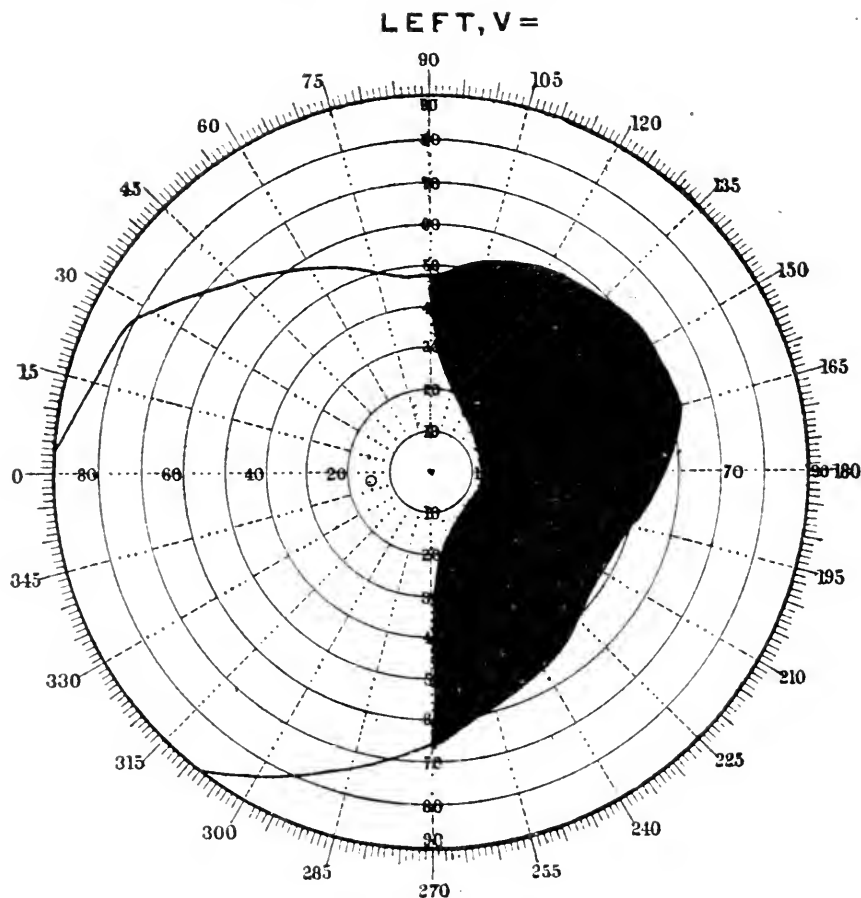
This patient was first seen in consultation with Dr. Bowers, July 16, 1894. In April she had had a spell of vertigo on rising in the morning and towards noon a second similar attack, and from this time on it had become almost impossible for her to name objects. Until the first of August she was seen five or six times, and careful examinations during this period showed neither anesthesia or paralysis. Ophthalmological examinations showed no optic neuritis, but an irregular left lateral homonymous hemianopsia.

The following report was received from the ophthalmological department of the Philadelphia Polyclinic: O. D., media clear, hyperopia 3 D., disc slightly red; O. S., media clear; small brilliant white spot, one-half diameter down and out from the disc; fundus otherwise normal in both eyes; good reaction to light on either half of either retina. With + 3.25 D., vision is at least as good as given above, but many letters are miscalled, and this happens as frequently with large as with small letters. Ocular movements are normal. The fields show hemiambyopia homonymous; right one-half impaired, blind passing  $10^{\circ}$  around fixation point and not reaching the median line above to  $30^{\circ}$ .



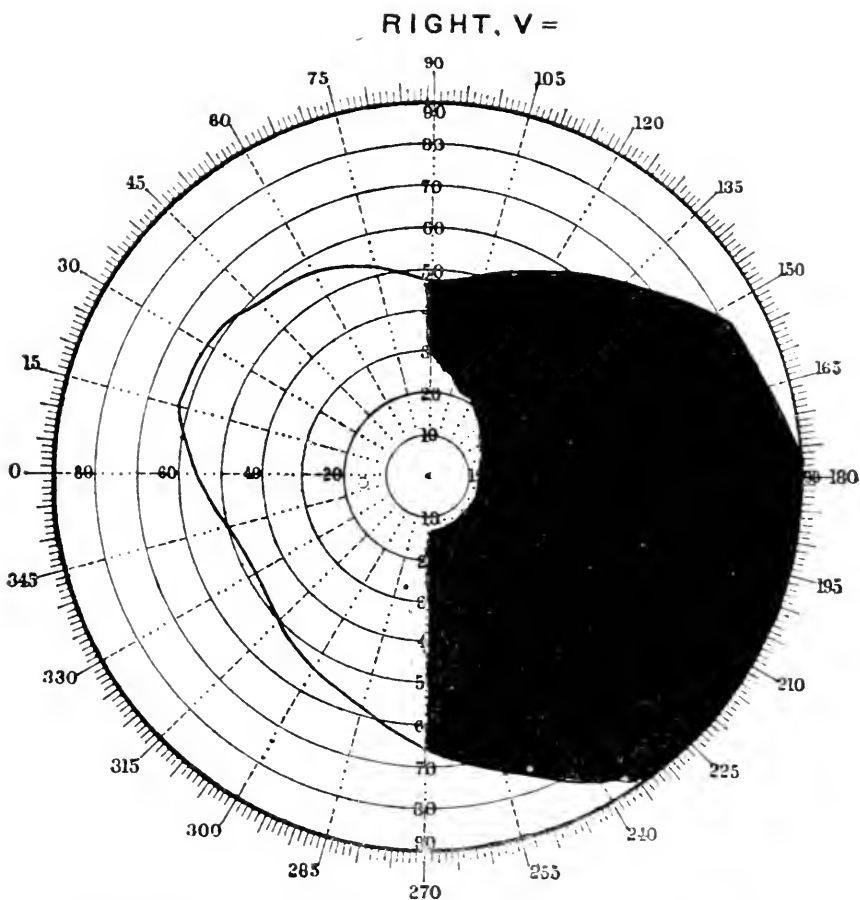
Name written by patient with verbal amnesia and partial word-blindness from lesion of the naming centre and adjoining regions.

She was word blind in large part but not letter blind and could name single letters slowly. A facsimile of her effort to write her name, "Margaret L.," is given.



Fields of vision in case of verbal amnesia with lesion of the naming centre and adjoining region.

She could not name objects either from sight or touch. When a pencil, pen, scissors, or purse was held before her, or when she was allowed to touch them she could not give their names, although she evidently understood what they were. On one occasion she called the scissors, "what I sew with," and the purse "what I buy with." At times she became much worried and emotional because of this inability to name objects. When such objects were named to her she would promptly and with evidence of satisfaction indicate that the names were



Fields of vision in case of verbal amnesia with lesion of the naming centre and adjoining centre.

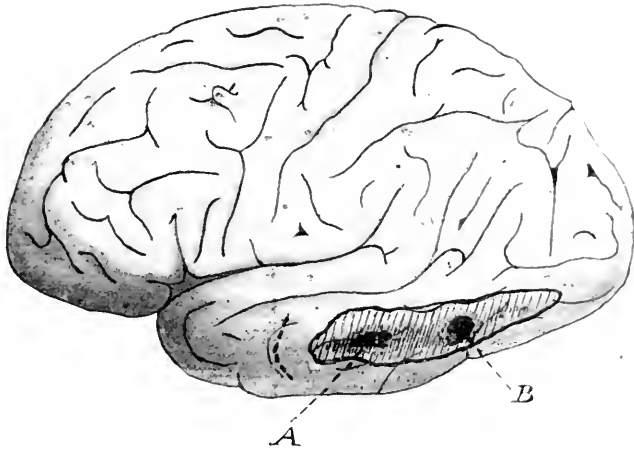
correct; and she could also, as a rule, repeat the names when spoken before her, but not always quickly, and occasionally she had considerable difficulty in repeating them. When asked her first name she said "Margaret" with facility, but she had great difficulty in mentioning her last name unless it was repeated to her, although she sometimes succeeded without this prompting. She used "yes" and "no" properly, and in many ways indicated that she knew what the objects were and their uses, but could not give their names. She talked spontan-

ously, but not freely, rarely using concrete nouns, and sometimes misplacing words. She was not seen by the reporters after August 1st, but Dr. Bowers has kindly furnished the notes of her condition from this date until her death.

August 1st she became unable to perform shoulder and elbow movements, and leg and thigh movements on the right side, but retained power in the hand and fingers, foot and toes. Gradually the paralysis of the right limbs became complete, but without loss of sensation. The skin on the paralyzed side assumed a pinkish hue and its temperature was increased. The pain in the head disappeared when the paralysis became complete, but returned to some extent a week before her death. Three weeks before her death she regained some power in the toes and fingers. During the last weeks of her life "yes" and "no" were the only expressions used by her. She became somnolent and, later, stuporous; she developed bedsores and lost control over both the vesical and rectal sphincters. Death occurred September 10, 1894.

An autopsy was made forty-eight hours after death by Drs. C. W. Burr, J. W. McConnell, W. Bowers and C. K. Mills. In the course of the removal of the brain, a small nodulated, half disintegrated mass, about the size of a hickory nut, was pulled out of the brain surface at a position which corresponds to the posterior fourth of the third temporal convolution, at B in Fig. (p. 7). The surface of the third temporal in its posterior half, to a much less extent the second temporal in the same general region, the fourth temporal presented a granular, slightly disintegrated appearance. On cutting into the temporal lobe a hard tumor, yellowish brown in color, was revealed; its hardest and apparently oldest part was at A, about the middle of the third temporal (medi-temporal) convolution, and passing slightly into the second temporal. The mass extended cephalad and caudad a short distance almost entirely in the white matter of the third temporal gyri, and a soft nodulated, disintegrated, more or less hæmorrhagic condition extended still further reaching caudad as far as the white matter of the middle of the occipital lobe, and cephalad to the junction of the first and middle thirds of the second and third temporal convolutions, as indicated by the dotted line. The parts chiefly destroyed were the white matter of the third, to a less extent of the second, and to a still less extent of the fourth, temporal convolutions. Internally the roof of

the posterior horn presented a slightly congested and granular appearance. The disease almost certainly started in the third temporal convolution, at a point with the posterior extremity of the horizontal branch of the Sylvian fissure. Microscopical examination by Dr. Burr showed the tumor to be a glioma.



Tumor of the third temporal convolution indicating the position of the naming centre: A, densest and probably oldest portion of the growth; B, place where the nodular mass was torn away during the autopsy; the heavier shading indicates the region of the greatest cortical and subcortical destruction.

**Hypothermia in the Insane.**—J. B. Bouchand (*Ann. Med. Psych.*, No. 2, 1894) reports thirty cases of lowered temperature among the insane, due to the feeble resistance and to depressing influences that exist in all the functions of the nervous system when the latter is diseased. Calorification is one of the functions of the nervous system, and subject to modifications like all the others. Hypothermia, then, this observer considers to be a state of inhibition in persons whose nervous system is profoundly weakened.

BRYSON.

# LOCOMOTOR ATAXIA: SUDDEN ONSET AND UNUSUAL SYMPTOMS; POSTERIOR SCLEROSIS OF SIMILAR SYMPTOMATOLOGY IN PATIENT'S WIFE.<sup>1</sup>

By F. SAVARY PEARCE, M.D.,

Philadelphia.

**D**R. S. WEIR MITCHELL kindly permits me to present the following unique case:

Mr. J. W., æt. fifty-one years, gives a history of a generally robust ancestry. Especially has no nervous diseases existed among any of his relations, excepting that of his wife, who suffers from advanced symptoms of locomotor ataxia, she being of no consanguinity, however. Patient denies having ever had venereal disease. He has had no recent acute illness, and has been in business as a miller continuously for many years. For the past twenty years he has had much care in nursing his invalid wife, but considered himself in perfect health until thirty days ago. At that time (October 26, 1894), about 3:30 p. m., after working at his accounts, he walked to the office door in perfect health, could see perfectly, but there was suddenly a slight sense of vertigo developed. Five minutes later he as suddenly developed double vision without other untoward subjective symptoms. There was no shock to the nerve centres, no headache, but slight dizziness, while his locomotion remained normal. He walked out into the mill, saw each workman double, etc., so much so that it became very confusing, and he closed one eye for relief. He drove then to his home two miles away. Says whenever he opened both eyes while driving along the road he saw two horses instead of the one ahead, this being the only confusion; and he felt no abnormality so long as one eye remained closed. He put his horse in the barn without difficulty, and he did not think the double images had grown at all farther apart. In fact, the diplopia has remained the same from the

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<sup>1</sup> Reported at the meeting of the Philadelphia Neurological Society, November 26, 1894.



onset up to the present time. The next day he drove some four miles on business, and incidentally saw his physician, who suggested that he should consult an oculist if the condition did not improve.

October 28th, the second day after the sudden onset, the patient felt a sense of numbness about the rectum when using toilet paper. There was no true anæsthesia. He also noticed this same paræsthesia on the scrotum, and extending to the glans penis, but nowhere else. Bowels and bladder were controlled normally.

October 29, Dr. Ramsay, of Chambersburg, Pa., examined the eyes and reported some trouble with the left optic nerve and muscular weakness.

On October 31 the patient became "weak in the knees," and for the first time experienced some awkwardness in walking. No new symptoms developed until five days later, when at eight in the evening of November 5, when about to retire for the night, a black spot appeared before his right eye which he could not wipe away. Presently all grew dark before both eyes. He lay down five minutes later. This was followed by a sensation of heat and quivering over a small spot in nape of neck. The patient's pulse became very rapid, so that he could not count it. Soon he began to "quiver and jerk from head to foot," and describes it as a "most miserable feeling." Thought his breath was "stopping," and there was a sense of pressure about the heart. This state of affairs continued for about five minutes. Suddenly his pulse came down to 95 per minute.

Twenty minutes later a similar attack occurred. One hour from this a third attack recurred, but of much less severity than the foregoing ones. Patient felt no pain afterward, but was uneasy and slept none that night. Next morning could walk, but was very weak and felt like "toppling over." Walking on bricks felt like treading on carpet, and stepping on the carpet felt like walking on soft pillows. (This has persisted to the present time.) Felt numb in left foot the same day (November 6).

The next day (November 7, 1894) the toes of right foot and fingers of left hand likewise became numb, but not so markedly as the left foot and leg to the knee. On November 8 his right hand became numb. Patient did not remark voluntarily that he experienced greater difficulty in walking in dark than in light, but on questioning thinks he can get along quite as well at night as in the day.

time. He becomes "numb" when he sits long, and striking his thighs produces a tingling sensation all through his body. Exercise relieves the numbness and tingling. November 20, for the first time, at 8 a.m., did he experience any true pain like a "crisis," at which time he felt a rather sharp twinge darting in left hypochondriac region and in the calf of the left leg. Has had a recurrence of this several times since at night, and of rather greater severity.

*Status Præsens.*—Good physique, muscles firm. No local deformities, atrophies, or tender spots on any part of the body. Lungs, liver, spleen and other organs normal, except that the second aortic sound of heart is clangy and pulse tension is somewhat increased. Controls bowels and urine; the latter is normal, excepting for excess of urates in the evening discharge. The patient has not lost weight.

*Nervous System.*—Station is fair with eyes open, but he sways visibly when eyes are closed. Knee-jerks absent and not reinforcible. No ankle clonus or tendo Achillis reflex. Muscle-jerks below the knees are lessened, abdominal and cremasteric reflexes present. Jaw-jerk present. Elbow-jerks are increased; shoulder, chest, and arm muscle-jerks are a little quicker in response than normal perhaps. Sensation to touch, pain, and thermal sense everywhere normal. Hands are not noticeably ataxic. The gait is unsteady and he feels the necessity of carrying a cane. The eyes were examined by Dr. Thompson, who reports as follows: Pupils react normally to light and accommodation. Fundus and discs normal, H = 1.50 D. Form and color fields normal. Paralysis of both external recti muscles.

*Conclusions.*—The case is interesting as an aberrant form of locomotor ataxia, rapid in its onset, with absence of Argyll-Robertson pupil, with an unusual ocular disturbance as the initial symptom, with the absence of marked ataxia, and the insular (as it were) singling out of areas of paresthesia due to exudate outside of, or to an acute subtle inflammation about the posterior roots of the cord.

The other point of interest is that the man's wife has posterior sclerosis, coming on first by diplopia twenty years ago; that three months later the double vision left her suddenly, that the right leg remained cold, and that six months after the incipency of the disease the wife suffered from a crisis of pain, which recurred again two

months later; that one year after the initial symptom, too, in the wife's case, she had the first sensation of weakness in the legs, which progressed rapidly, and that she has been unable to walk alone for sixteen years; and finally, that her eyes have remained quite normal ever since she lost the double vision, the right eye remaining more acute of vision than the left.

Our patient is good enough to come here to-night for your examination.

Mr. J. W. is getting massage daily, Bichloride of Mercury grain  $\frac{1}{10}$  three times daily; has received four treatments of hypodermic injections of Brown-Séquard (III X) fluid, and says he feels somewhat rejuvenated, and has better co-ordination already. A month later the good result of this therapeutic treatment can be more definitely decided. I have seen good results from its use in one other case of posterior sclerosis.

Dr. Mitchell is of the opinion that early vertigo is often a precursor of a giving out of the ocular balance, as in this case. He has now seen four instances where husband and wife suffered from posterior sclerosis.

In these two cases here reported, syphilis is ruled out of consideration as an etiological factor. In the family record the syphilitic taint was excluded in a second case, the husband developing the disease five years after the wife's symptoms began. In the other two families, where both husband and wife developed posterior sclerosis, there was a specific history on the husband's side in one case, and on the wife's side in the other, with frequent cohabitations in both.

Thanks are hereby extended to Drs. Noble and Critzman, who referred to the case and aided in making up the history of the onset of the initial symptoms.

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**A Case of Pseudo-Bulbar Paralysis.**—Professor Filatoff presents a child of eleven years whose parents had noticed a gradual increase in the abdomen and difficulty in pronouncing words. On close examination the spleen was found enlarged, other internal organs normal. The voice had a nasal twang, and from time to time there occurred oscillatory movements of the eyeballs. The inferior branch of the facial is paralyzed, the masseters functionate well, but the pterygoids are affected. The tongue is motionless, but not atrophied. The soft palate is discolored and anæsthetic. The electrical irritability of the muscles supplied by the facial nerve and the muscles about the throat are normal. Speech is very difficult, deglutition not difficult and the muscles of the extremities are rigid but not atrophied. Administration of potassium iodide, one gramme daily, has produced no results.

W. C. K.

# THE HISTOLOGICAL CONFORMATION OF THE MEDULLA.<sup>1</sup>

By WILLIAM C. KRAUSS, M. D., F. R. M. S.,

Professor of Pathology in the Medical Department of Niagara University, Buffalo, New York; Neurologist to the Erie County Hospital, etc.

THE study of the transition of the cord to the brain is perhaps the most difficult task in the anatomy and histology of the nervous system. To trace the origin and direction of nerve bundles, the appearance of new masses of gray matter, and the coalition of different tracts requires much time, excellent specimens, correct drawings, and careful explanations. The discovery of the Weigert method of staining and the Pal modification has proved of great benefit in following these complicated changes, and has permitted the investigator to trace the course of the different tracts with much satisfaction and success.

The internal configuration of the spinal cord from the third or fourth lumbar segment to the second or third cervical is practically the same. Slight changes occur characteristic of the lumbar, thoracic, and cervical regions, such as the development of the dorsal cornua or lateral cornua or of the ventral cornua; but these are of minor importance when compared to the changes occurring between the first and second cervical segment to the beginning of the pons.

The spinal cord within the limits referred to consists of a cylindrical mass of nerve elements held together by neuroglia cells and encased in appropriate membranes, lending support, protection, and nutrition. The white matter, composed of medullated nerve fibres, is arranged peripherally, while the gray matter containing the ganglion cells is situated in the interior. The gray matter consists of two crescentic masses, their convex surfaces facing and connected together by a bridge of gray matter, the commissure; the whole resembling very much

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<sup>1</sup> Illustrated by a series of six slides, to which was awarded the cash prize of the American Microscopical Society, held at Madison, Wisconsin, August 19-21, 1893. Mounted and photomicrographed by the author. See frontispiece.

the letter H. The gray matter is divided arbitrarily into dorsal, lateral, and ventral cornua, and the gray commissure inclosing the spinal canal. From the dorsal and ventral cornua bundles of nerve fibres pass through the white matter to the periphery of the cord, forming the dorsal and ventral spinal roots.

The white matter is divided into two hemispheres by the dorsal and ventral fissures, and each hemisphere is separated into dorsal, lateral, and ventral columns. These columns are further subdivided as follows: The ventral, into the columns of Turck and ventral columns proper; the lateral, into the pyramidal tracts, cerebellar tracts (Flechsig), and tracts of Gowers; the dorsal, into the columns of Goll, columns of Burdach, and Spitzka-Lissauer tracts. These subdivisions are mapped out according to the part they play in conducting nerve impulses to or from the brain centres. The continuation of the cord caudad is the cauda equina, cephalad, the medulla.

From about the middle of the thoracic region fibres pass from the pyramidal tracts in the lateral columns through the base of the ventral cornua to the ventral columns of the opposite side. This decussation is scarcely recognizable at first, but gradually increases cephalad until the first cervical segment, when the remaining fibres not yet decussated pass to the opposite side in bundles, severing the ventral cornua from the central gray matter and pushing them laterad. The lateral cornua become prominent, and from their cells originate the XI. pair of nerves (spinal accessory), passing through the white matter and emerging at the side of the medulla between the dorsal and ventral roots.

While these changes are going on in the ventral and lateral columns the dorsal columns increase in size, the funiculi graciles (tracts of Goll) and funiculi cuneati (tracts of Burdach) develop, pushing the dorsal cornua before them. The long, slender bodies of the dorsal cornua swell, enlarge, become rounded, and are connected to the central gray matter by thin pedicles. The substantia gelatinosa Rolandi becomes prominent, and medullated nerve fibres arise adjacent and ectad passing cephalad as the ascending branch of the Trigemini. The formatio reticularis develops in the lateral columns, appropriating to itself the severed portions of the ventral cornua, except some small aggregations of nerve cells surrounded by patches of gray matter, the most import-

ant of which is called *nucleus lateralis medius*, or *nucleus ambiguus*.

Further cephalad there arise in the *funiculi graciles* and *cuneati* small masses of gray matter containing isolated groups of ganglion cells, designated *nuclei funiculi graciles* and *nuclei funiculi cuneati*. The decussation of the pyramidal fibres being completed and the pyramids fully developed, other fibres may be observed coming from the former dorsal columns, passing concentrically about the central canal and decussating ventrad of it, then arranging themselves on both sides of the ventral fissure dorsad of the pyramids forming the *lemniscus*.

The continuance of these fibres, now designated *fibræ arcuatæ internæ*, decussate in the median line from the central canal to the ventral fissure forming the *raphe*.

In the lateral regions immediately dorsad of the pyramids small masses of ganglion cells, symmetrically arranged, begin to appear as intestinal-like masses, called the *olivary bodies*. Generally there are two or three of these bodies present besides the principal ones, and to these have been given the names *internal* and *external accessory olivary processes*.

The central canal, at first circular, becomes elliptical, and traced cephalad it widens out, rises to the dorsal surface, and terminates in the *calamus scriptorius*, then into the fourth ventricle. In the base of the ventral cornua, a group of multipolar ganglion cells, appear the *nucleus* of the XII. pair or *hypoglossal nerves*. The nerve fibres coming from these cells pass ventrad through the *formatio reticularis*, between the *olivary bodies* and the pyramids, and emerge exteriorly laterad of the pyramids. In the base of the dorsal cornua, now pushed latero-dorsad, and appearing as the gray matter in the floor of the fourth ventricle, appear masses of small ganglionic cells, the *nuclei* of the X., IX. and VIII. pairs of nerves.

The *cerebellar tracts*, or tracts of *Flechsig*, have joined with other fibres coming from the dorsal and lateral columns, and pass cephalad into the cerebellum as the *corpora restiforme*.

In the floor of the fourth ventricle, close beside the median *raphe*, a small group of ganglion cells may be observed, the *nucleus funiculi teretis*. Ventrad of these cells an oval bundle of nerve fibres passing cephalad may be seen, called the *fasiculus longitudinal posterior*.

The next important change is the appearance of the transverse bundles of fibres forming the pons, and with the appearance of these fibres the medulla ceases its existence.

#### METHODS EMPLOYED IN HARDENING, STAINING, MOUNTING AND PHOTOGRAPHING THE SECTIONS.

The medulla was hardened in a five per cent. solution of potassium bichromate for six weeks, the fluid having been frequently renewed, then dehydrated and decolorized in alcohol, repeatedly changing the alcohol, until it became slightly tinged. The medulla was then transected into six pieces, imbedded in Schiefferdecker's celloidin solution, then fastened upon corks, preparatory for section-cutting.

The staining method used was the Pal modification of Weigert's method.<sup>2</sup> The sections were placed in Weigert's staining fluid, to which had been previously added three to five drops of a saturated solution of lithium carbonate for every ten cubic centimetres of the stain used.

	0.75-I.O.	part hæmatoxylin.
90	"	water.
10	"	alcohol.
1	"	lithium carbonate.

After five or six hours they were removed and carefully washed in water to which some of the carbonate of lithium solution had been added. They were then placed for ten or fifteen seconds in a one-fourth per cent. solution of the permanganate of potash, rinsed in thirty per cent. alcohol, and placed in the differentiating bath.

Oxalic acid,	1	part.
Sulphite of soda,	1	"
Distilled water,	200	"

The sections were placed in the bath singly, and removed as soon as the differentiation between the white and gray matter was very patent. They were then washed repeatedly in water, and allowed to stand in water for twenty-four hours. After dehydrating in

<sup>2</sup> Wiener Medizinische Jahrbücher, 1887, p. 580. Author's paper on "Some Methods of Treating Nerve Tissues." Proceedings of the American Society of Microscopists, 1890, pp. 116-120.

strong alcohol they were cleared in Weigert's clearing mixture.

Xylol, 3 parts.

Carbolic acid, 1 "

Sulphate of copper, enough to cover the bottom of the vessel—then mounted in balsam.

By this method the medullary nerve fibres were stained light blue, the neuroglia, connective tissue and vessel walls were rendered white or yellowish white, while the ganglion cells became transparent.

No. 5 was double stained in picro-carmin in order to bring the nuclei of the X. and XII. pairs of nerves into prominence. The method employed was to place the sections in picro-carmin after their development had been arrested by the water bath, then washed, dehydrated and mounted in the ordinary way.

The sections were photographed by means of an ordinary camera, the lens having been removed and the tube of the microscope fitted with a movable diaphragm inserted into this opening. A Spencer and Smith one-inch aplanatic ocular and a Swift three-inch objective were employed for all the sections except the fifth, where a Spencer four-inch objective was used.

Direct sunlight furnished the illumination; and exposures of three to five seconds were allowed on Carbutt's orthochromatic plates, sensitometer No. 27. The negatives were developed with eikonogen fixed in the alum and hyposulphide of soda solution, printed on albumen paper and toned with the chloride of gold solution.

The plate does not serve as an index for the time spent in trying to produce a photograph from a blue and white field. By careful manipulation of the mirror and substage, I was able after repeated failures to get a sufficient contrast between the blue and white so as to enable the engraver to perform his work satisfactorily.

FIG. I.—Transection of the medulla at its junction with the cord (first cervical segment), showing the decussation of the pyramidal tracts and formation of the pyramids. In the dorsal columns may be seen the newly-formed funiculi cuneati and graciles, and in the lateral columns the origin of the fibres of the ascending branch of the trigeminus nerve. Magnified  $2\frac{1}{2}$  diameters.

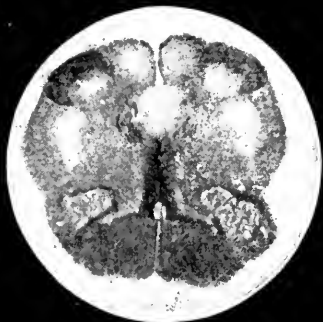




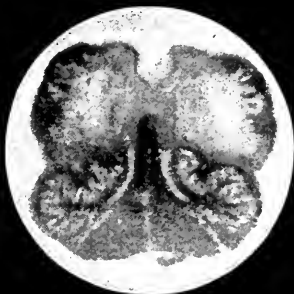
I



II



III



IV



V



VI



FIG. II.—Transection of the medulla one-half centimetre cephalad of Fig. I., showing the decussation of fibres (sensory) coming from the funiculi cuneati and graciles, forming the lemniscus. Lateral of the lemniscus appear the olivary bodies. Magnified two diameters.

FIG. III.—Transection of the medulla one-half centimetre cephalad of Fig. II., showing the fibræ arcuate internæ decussating and forming the raphe. The olivary bodies are more developed, and the spinal canal may be seen to gradually ascend to the dorsal surface, where, further cephalad, it terminates in the calamus scriptorius, then into the fourth ventricle. Magnified  $2\frac{1}{2}$  diameters.

FIG. IV.—Transection of the medulla one-half centimetre cephalad of Fig. III., showing the calamus scriptorius. The olivary bodies and the accessory olivary bodies are now plainly visible. Magnified two diameters.

FIG. V.—Transection of the medulla one centimetre caudad of the pons, showing the floor of the fourth ventricle, the development of the restiform bodies, the fibres of the hypoglossal nerves separating the substantia reticularis alba from the substantia reticularis grisea, and the ascending branches of the glosso-pharyngeal nerves. Magnified  $1\frac{1}{4}$  diameters.

FIG. VI.—Transection of the medulla one-half centimetre caudad of the pons, showing the fasciculus longitudinalis posterior in the floor of the fourth ventricle and indistinctly the emergence of the fibres of the vagus nerves. Magnified  $1\frac{3}{4}$  diameters.

The nuclei of the XII, XI, X, IX and VIII pairs of cranial nerves are situated in the medulla embraced by these sections, but on account of their being rendered transparent by the Pal method, cannot be distinguished.

## NOTE OF A CASE OF SOFTENING OF THE RIGHT ANGULAR GYRUS, WITH LEFT- SIDED PTOSIS.

BY C. A. HERTER, M.D.,

Visiting Physician to the City (Charity) Hospital; Consulting Physician to the Babies' Hospital; Professor of the Anatomy and Pathology of the Nervous System, New York Polyclinic.

THE following case deserves to be placed on record, because it illustrates the production of ptosis upon one side by a limited cortical lesion in the opposite hemisphere :

A man, aged about sixty, very much emaciated, was admitted to the City (Charity) Hospital in a state of stupor. He was under observation two and one-half days, during which time the temperature ranged from 100° to 102°. There were some signs of tuberculosis of the lungs, there was a bad cystitis, the heart was very weak and rapid, and the radial pulse could not be felt most of the time that the patient remained under observation. The right arm and the right leg were quite flacid, and were only slightly moved on painful stimulation. The face did not seem to be involved. No weakness could be detected on left side of body. Both knee-jerks were lost. There was no rigidity anywhere. On the left side there was ptosis not quite complete. The left pupil was slightly dilated, and reacted less well to light than the right pupil.

Although the weakness on the right side was not considerable, it seemed sufficiently pronounced to justify the diagnosis of a crossed hemiplegia. The nature of the lesion was thought to be either softening of the pons from atheroma or tubercular meningitis with multiple cheesy masses.

Death occurred from suppurative nephritis and exhaustion. The autopsy showed the existence of extensive pulmonary tuberculosis with cavities, and pyelitis, with suppurative nephritis. In the right hemisphere, just below the interparietal fissure, was a circular patch of softening, one inch in diameter, occupying the angu-

lar convolution. The softening involved the cortex, and, to a slight extent, the white substance beneath it. The position of the area of softening is shown by the accompanying diagram. It is safe to refer the left-sided ptosis to the lesion in the right hemisphere. The right-sided hemiparesis cannot be satisfactorily explained, but may have been due to a uræmic condition resulting from the kidney disease.

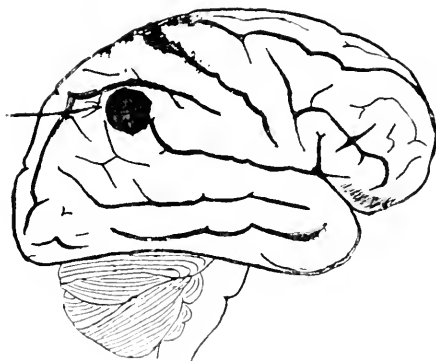


Diagram illustrating position of area of softening in right hemisphere.

**Primary Menstrual Psychosis.**—Freedmann (*Münch. Med. Woch.*, No. 1, 1894), describes a periodic mental disorder that appears at the beginning of puberty and which disappears when the menstruation is well-established. It should not be confounded with the periodic menstrual psychosis already described, but must be separated from it under the name of the menstrual psychosis of development. It bears but slight analogy to other disorders of puberty. In principle, however, it may be compared to those single attacks of transitory overthrow of consciousness that sometimes occur suddenly at the time of puberty. The character of the difficulty is identically that of the very large group of psychosis of somatic origin. The normal mind possesses extraordinary force of resistance to pernicious influences, and an astonishing recuperative power. Psychosis due to external influences and remaining functional, do not survive the conditions that produced them. They do not explode except under pressure of pathological stasis. When there is defect in recuperative power, a chronic psychosis results. If the defect is one of resistance merely, there is a periodic psychosis of brief duration. Transient psychic abnormalities are no more due to hysteria and epilepsy than they are to other somatic and nervous causes. BRYSON.

## A CASE OF DIGITI MORTUI OF FOUR AND A HALF YEARS' DURATION.

By C. E. STANLEY, M.D.,

Middletown, Conn.

THE following case of *digiti mortui* has been of interest on account of its comparative rarity, long duration and resulting trophic changes:

M. C., female, single, aged thirty-five years, temperate, of slight build, feeble-minded. Physical defects: speech affected by a lisp; slight posterior curvature of spine (kyphosis). Head well and symmetrically developed. Imbecility is attributed by her friends to an attack of scarlet fever in childhood. She attended school, but is unable to read or write. Her father—whom she resembles—and her mother are both dead. Three brothers and two sisters survive, and are said to be healthy. When admitted to the Connecticut Hospital for the Insane, May, 1885, she had no delusions, but was subject to occasional brief paroxysms of irritability of temper, during which she indulged in loud, profane and abusive language. In the intervals she was sociable, quiet and industrious. The outbursts of passion were easily provoked and as easily allayed. A word of remonstrance often sufficed to divert her attention, when she would quickly regain her usual serenity, and frequently ask to be pardoned for disagreeable sayings she had uttered. She was fond of notice, emotional, hypersensitive and sometimes rather simple and childish in speech and actions. No history of gout, rheumatism or syphilis was elicited. The reflexes were normal, menstruation was regular; urine not examined.

On March 12, 1890, while out doors taking the prescribed morning walk, she experienced a peculiar sensation of numbness in one of her hands. The day was cold, but she was warmly clad and her hands were protected by woolen mittens. Upon investigation it was discovered that the second finger of the right hand was blanched, of a death-like palor, smooth, apparently bloodless, and in the condition known as local syncope or

dead finger. The attack, unaccompanied by constitutional symptoms, was sudden, painless and confined to one finger. There was loss of cutaneous sensibility in the affected part and its temperature was sensibly lowered. The finger felt as if dead. With the continued use of hot applications, after the lapse of about two hours the above symptoms gradually disappeared, and the part was restored to nearly its normal appearance. The finger, however, never quite regained its former vigor. Similar attacks, of longer or shorter duration, have since occurred at varying intervals in the same finger, and the corresponding one of the left hand. Cold was commonly the excitant, although undue mental excitement seemed at times to provoke an attack. The patient passed from observation in August, 1892, and was not seen again until August, 1894. At the latter date, four and a half years from its inception, the disease was still active. Other fingers, and, also, to a less degree, some of the toes had become similarly involved. The fingers first attacked by the disorder now presented a pale, shrunken appearance, with the joints abnormally prominent, the result, doubtless, of trophic changes in the phalanges.

The pathology of *digiti mortui* is still somewhat obscure. While some authors consider the affection as an abortive form of Raynaud's disease, and others as merely a phase of multiple neuritis, yet most recent writers describe it as a vaso-motor disturbance, and regard it as primarily an angio-spasm due to over excitation of the vaso-motor centre, followed by a neuritis. In the case narrated above the disease is associated with a psychosis as well as a hypersensitive condition of the nervous system. Nothing in its history, however, indicates the influence of any toxic material as a causative factor.

Treatment in the form of electricity and tonics has proved of no special benefit. The disease has been steadily and progressively increasing.

## Asylum Notes.

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BY RALPH WAIT PARSONS, M.D.

*Some Reasons Why More Original Investigation and More Literary Work is Not Done by the Physicians in Large Public Hospitals for the Insane, With a Suggestion of Certain Remedial Measures.*

Dr. S. Weir Mitchell's severe criticism, not only of the general management of the hospitals for the insane, but also as to the lack of thorough and scientific work done by the physicians connected with such institutions, has caused much thoughtful consideration. It is certain that a great deal more pathological and literary work should be done, but that there are many and serious difficulties in the way of pursuing careful, scientific study of the clinical and pathological material at their command, and of reporting the results thus obtained, there can be but little question. Dr. Hinckley, in his article which appeared in the JOURNAL OF NERVOUS AND MENTAL DISEASE, September, 1894, strongly points out how frequently political interference impedes the up-building of a scientific hospital for the insane. The superintendent is frequently requested to do many things which he, in his best judgment, or from a sense of equity and duty, feels he ought not to do. He shows how attendants and other employes, who have been deprived of their positions for disobedience and neglect of duty, have on several occasions been reinstated for purely political reasons. The damaging effects of political interference upon the management of our public hospitals for the insane is also frequently alluded to in the letters written in answer to Dr. Mitchell's circular letter.

Dr. Hinckley also calls attention to the fact that the financial resources at the superintendent's disposal for the advancement of the welfare of the institution under his care are frequently far too small, and that even the expenditure of a few dollars for minor surgical or lesser gynæcological operations is grudgingly allowed, and



requests to purchase apparatus for electro and hydro-therapy are positively refused. He further says, "The great misconception of the true principles of economy, and the fatal method of forcing down legitimate expenses in order to make an actual showing of dollars and cents, which is too often the policy adopted by the managers of our institutions throughout the land, are fraught with the most baneful consequences."

Dr. C. L. Dana says in his letter to Dr. Mitchell, "I believe that all over the country the great trouble is that the people do not understand what a difficult, costly and important problem the care of the insane is. Hence comes the evil of political management, and as a result the necessity of doing things economically and yet of taking care of the 'boys.'"

A great deal of the superintendent's time is taken up with purely executive work, the designing and overseeing the construction of new buildings, new roads, and the like, leaving him little time for scientific research, or for a systematic study of his cases. Dr. Billings, in speaking of the matter, says, "The energies of the well-qualified asylum superintendent have been heretofore largely devoted towards increased accommodations for the insane, and their duties as administrative officers, charged with the planning and erecting of new buildings and with the details of asylum management, have left them little time or opportunity for scientific investigations or for making themselves acquainted with what has been done by others in the diagnosis and treatment of insanity."

One great obstacle that the asylum physician finds to the careful and systematic study of his cases, or to the devoting of his attention to pathological research, is the immense amount of purely clerical work he is required to perform. The regulations of the State Commission in Lunacy require that a note shall be taken every day for ten days in the case of each newly-admitted patient; after this period one note every month during the first year, and after the first year one note is to be taken at least every three months as long as the patient remains under treatment. Of course, special notes are to be made concerning the progress of the case, or for other reasons, as the physician in charge of the service to which the patient belongs finds necessary. Now, suppose that in one department alone, say the male department, consisting of six hundred patients, thirty patients

are admitted per month, and suppose that one patient is admitted daily, we shall then have to make ten notes daily for the new admissions, besides those required concerning the mental and physical condition of the first or second preceding ten patients. Now, suppose that one-half the patients under treatment in the service have been admitted during the period of one year. We would then have to make ten notes a day besides those mentioned above, and every third month a note is to be taken of every patient in the service. It will be seen, then, that the senior physician in charge of a service of between five and six hundred patients has a great deal of writing to do in order to keep up the histories of his patients. The senior, as a rule, takes the history and makes the preliminary mental and physical examination of each newly-admitted patient, and, furthermore, he is required to make a record of the prescriptions which he has deemed necessary to order according to the daily needs of the patients under his charge. Much time, also, is often taken up in consulting with the friends of patients concerning the mental and physical condition of their relatives, and frequently they insist upon seeing and obtaining the opinion of the senior, although the other physicians connected with the service could, in most cases, answer all questions satisfactorily. After attending to various other matters of executive importance in relation to the daily management of his service, it is not difficult to understand that the senior assistant has little time left, after the regular daily routine work is over, for systematic study or pathological research.

Now, as to the assistant physicians connected with a service like the one already alluded to. A large proportion of their time is taken up with purely clerical work; that is, making duplicate medical certificates, copying certificates into the histories of patients, and copying notes taken by the senior physician concerning the condition of various patients; and it should be remembered that the notes regarding the recent cases are often very full, so that a large amount of copying is required in order to keep the histories of the patients up to date.

It will, as I think, be generally agreed that this vast amount of clerical work is of little value, as such, to the physicians whose duty it is to perform it. If an intelligent, conscientious clerk were employed to do this daily routine of office work, the physicians then would have a great deal more time to devote to their legitimate medi-

cal work, to study of the symptoms, course and results of treatment of mental diseases, and to work in the pathological laboratory.

It is urged that the histories of patients are private data, and thus that the physicians alone should do the copying in the case books. But, surely, an intelligent clerk, who has shown himself qualified to hold a position of responsibility and honor, will be no more likely to divulge professional secrets in relation to patients than a trained nurse, who is often well acquainted with private matters concerning the patient's life and personal character. As to the propriety of asylum physicians devoting so much valuable time to purely clerical work, which a person experienced in office routine and copying work could do just as well, or even better, allow me to quote from the letter written by Dr. Landon Carter Gray in response to Dr. Mitchell's circular letter. He writes: "Some means should be devised by which the physicians can devote a proper amount of time to the study of their cases from the standpoint of the physician and not of the hotel-keeper." Dr. Charles K. Mills in his letter also refers to this point in no uncertain manner. He writes: "The medical staff should be enlarged. Individualization in study and treatment should be more thorough. Physicians should be relieved from much executive and miscellaneous non-medical work, to which they are now compelled to give too much valuable time."

Another plan that might be adopted to reduce the amount of clerical work which the assistant physicians have to do, and at the same time encourage them to active scientific work, would be to divide a given service into much smaller divisions than is now done, and to more carefully classify the patients, making one small division of the recent admissions, another of the infirm, and larger divisions of the more chronic cases. The senior would have a special supervision of the recent admissions, with the aid of one of the juniors, and would also have a general supervision of all the other divisions; a junior, under the general supervision of the senior, having special supervision of each of the other divisions. At the expiration of stated periods of time the juniors would change from one division to another in rotation. Thus each physician would have only a moderate amount of writing to do, and no one of the junior assistants would have the advantage over the other members of the staff of being constantly con-

nected with an acute service, and other physicians the disadvantage of being in charge of divisions almost wholly made up of chronic cases. Beside this, each physician could then follow his cases much more fully and accurately, as they might be transferred from one division of the service to another. This plan could be carried out still further by the physicians in the male and female departments changing places, at certain stated intervals, in order to give each member of the staff experience in the manifestations of mental disease in both sexes.

Dr. B. Sachs is strongly of the opinion that the clerical matters and medical work should be separate, so that the physicians might come in much closer personal contact with their patients, and thus be enabled to study their mental peculiarities more thoroughly. He says: "I am a strong believer in the good which results from intimate personal contact between the physician and the insane patient." He even goes so far as to recommend that there should be one physician to from ten to twenty patients. This would certainly be of great advantage to both physicians and patients, but would be impracticable in our large public hospitals for the insane. Dr. W. H. Draper writes in this connection: "Reform also is needed in the administration of our asylums, which should provide a separate organization for carrying on the executive and medical departments."

Another reason why, as I believe, more original work is not done by asylum physicians is, that they lack the stimulus of coming in frequent contact with specialists and general practitioners in the outside world. Many of our hospitals for the insane do not have a consulting staff. There can be no question that great benefit would be derived from having a consulting staff connected with each hospital for the insane. The great drawback is, that many of the large hospitals are located at a considerable distance from the great medical centres, and it is doubtful whether busy practitioners and specialists would feel that they could spare the time, if only for a few hours, six or eight times a year, to go to the hospital in the function of consultants. Several years ago a consulting staff was connected with the Hudson River State Hospital, but after a time the visits of the consultants ceased altogether. It would seem, then, that the specialists and other eminent practitioners ought to make some concessions on their part as to time and conven-

ience toward the advancement of the clinical and pathological study of mental diseases.

Another important drawback to the close and thorough study of clinical material in our public hospitals for the insane is the small number and inferior quality of attendants, for it is a well-known fact that physicians are often much aided in their study of cases of mental disease by the careful description of their words and actions given by an intelligent and observant attendant. Many insane patients have considerable voluntary control over their intellectual processes, and knowing that the physician is the judge as to their sanity or insanity, will control their words and actions in his presence, but as soon as he has left the ward will freely manifest these evidences of insanity in the presence of their attendants.

In many of our State and county institutions the attendants are drawn from the less intelligent classes of society, and are often coarse and clumsy in their ways, and some of them are even stupid. They may have been day-laborers, factory hands, farmers, and, in general, men "out of a job." The women have done housework, are often illiterate, and have only a faint idea, if any, as to the responsibility connected with the position which they seek to fill. These persons often secure and retain their positions as attendants through political influence, although they may be wholly unfitted for their positions. Dr. Hinckley, in his article already referred to, in speaking of political influence in our public asylums, says: "Positions of grave responsibility are filled by employees whose stupidity, illiteracy and total lack of adaptability are painfully apparent."

It has been the general experience of medical superintendents of hospitals for the insane, however, that nurses who have been originally trained in general hospitals are not well adapted to the work of caring for the insane. The best nurses for this class of work are those who, having the proper mental and physical qualifications, have received their original training in a hospital for the insane. The pecuniary and other considerations, such as accommodations, food, and opportunity for learning, which they receive, should be such as to induce a good class of men and women to take and hold these positions.

It is certain that every hospital for the insane should have a training school connected with it. This would

be a great advantage to the institution, the physicians and the patients. The attendants should have a systematic course of didactic and clinical lectures on the elementary principles of anatomy, physiology, surgery, therapeutics and psychiatry concerning the care, management and nursing of patients suffering from mental and nervous diseases, and in general nursing. Through the medium of a well-organized training school a great advance in the study and treatment of insanity could be made. But, under existing circumstances, it is very discouraging to endeavor to instruct attendants, many of whom are illiterate and who take little interest in their work, and who remain in the service of the hospital only a few months, and then resign their positions to engage in some other occupation.

To make this point more clear. I resigned my position on the staff of the Hudson River State Hospital about a year ago. At that time there were connected with the male department between seventy and eighty attendants. On a recent visit to that institution I observed that there were not more than six or eight attendants connected with the male service who had been there a year ago, and of these not more than three or four were connected with the hospital when I entered the State service in 1890. I believe that the constant changes in the corps of attendants, resulting from causes already mentioned, are pernicious, both to the interests of the institution and to the interests of the patients. Not only would the comfort and welfare of the insane be promoted, but I feel sure that the total percentage of recoveries would be increased through the services and co-operation of experienced attendants. The intellectual and moral control that intelligent attendants are often able to exert over their patients is large, and thus many cases of insanity are favorably influenced by the power of the stronger mind over the weaker.

In conclusion, I would sum up as follows, to wit :

1. The superintendent should be relieved of much of the executive and non-medical work which he is now called upon to perform. In order to obtain this object each large public hospital for the insane should have an assistant medical superintendent, who will share the executive work with the superintendent so much that each may have time to devote to the study of their cases and to the advancement of the work in the domain of psychiatry; and furthermore, they would thus stimulate

their assistants to the careful and accurate study of the phenomena of mental and nervous diseases, and to thorough pathological investigation.

2. A sufficient sum of money should be appropriated each year, which the superintendent can have at his disposal for the purchase of instruments for laboratory work and for scientific and therapeutic purposes.

3. The assistant physicians should be relieved, as far as possible, from the large amount of purely clerical work they now have to perform by either providing a clerk for the purpose or by increasing the staff so that the non-medical work assigned to each physician might be reduced to a minimum, thus allowing much more time for the systematic study of their patients and for pathological and literary work.

4. Men and women of intelligence and with the necessary qualifications should be appointed to fill positions as attendants, without reference to politics; and they should be given a sufficient salary, in order that it may be to their interest to hold their positions and thereby promote the interests of the institution and the welfare of the patients consigned to its care.

5. Every hospital for the insane should have an attendants' training school connected with it, in order that the best results of treatment may be obtained.

**Contribution to the Etiology of Facial Paralysis.**—Dr. Francisco Dominguez y Raldan, after making a careful study of facia, paralysis, in the *Anales de la Real Academia*, session of April 22, 1894, makes the following assertions :

1. The facial nerve, after the exit from the stylo mastoid foramen, does not penetrate into the parotid gland.

2. The facial nerve extends a distance of two centimeters in the space called infundibulum stylo-mastoidian.

3. The superior part of the infundibulum is formed by the widening of the Fallopien conduit. The periosteum covering it is continuous with that which covers the mastoid apophysis and the osseous portion of the external auditory meatus.

4. In this part of the infundibulum the facial nerve is surrounded by a very dense cellular tissue and by the lymphatic space described by Rudinger.

5. Inflammations of the external auditory meatus may provoke an inflammation of the periosteum covering the osseous portion of this meatus.

6. The periostitis, which takes its origin in the external auditory meatus, may propagate itself to the stylo-mastoid periosteum, provoking a facial paralysis by compression or alteration of this nerve in the superior part of the stylo-mastoid foramen.

7. The function of the chorda-tympani not being disturbed, may help to localize the trouble in the position above indicated. KRAUSS.

## Periscope.

### PATHOLOGICAL.

**Contribution to the Study of Syphilis of the Nervous System.**—F. Raymond (*Archives de Neurologie*, No. 83, 1894) records three cases of interest, and gives anatomical and pathological details of value. The first case showed diffuse vascular lesions of the cerebral cortex, the direct continuation of gummatous lesions of the large vessels; the second likewise presented vascular lesions, but they were limited to the cortex; in the third there was a similar irritative process, but more circumscribed in its vascular areas, and existing in the smallest capillaries only. In these three cases, the cerebral lesion was also associated with diffuse lesions of the spinal cord, evidently syphilitic. The first two cases were syphilitics, who became parietic. Raymond looks upon general paresis of syphilitic origin as a diffuse vascular encephalitis. But all subjects of diffuse vascular encephalitis are not syphilitic. General paresis supervenes only when nervous elements begin to suffer from defects in the apparatus of nutrition. The period of resistance may be very long and symptoms not well marked in consequence. But at a certain moment the nerve-tissue gives way; and the encephalitis, up to this point purely vascular, becomes mixed: elements of the neuroglia hypertrophy become sclerosed, nerve tubes are destroyed, beginning with the superficial ones of the convolutions, nerve cells undergo a change, and the injury to the cortex is now permanent and irremediable. Beside the specific agent, heredity must also be considered as a factor in the weakness or stability of nerve elements. Certain syphilitics can stand an interstitial encephalitis for a long time without becoming parietic; they may even recover if nerve tissue remains intact. Others, again, fall quickly into dementia, because their nervous structure possesses no power of resistance. BRYSON.

**Pathological Anatomy of Multiple Sclerosis.**—Dr. E. W. Taylor, of Boston (*Deutsche Zeitschrift für Nervenheilkunde*, V. B. I.). The paper comprises a report of three cases of multiple sclerosis of ordinary clinical course, with careful microscopical examination.

The points of special interest in the various cases are: (a) The unusual extent of the pathological process at several levels, involving a complete transverse degeneration of the cord. (b) The equal affection of gray and white matter. (c) The degeneration of anterior and posterior roots. (d) The involvement of the nuclei of all the cranial nerves in medulla pons and region of corpora quadrigemina. (e) A patch of continuous sclerosis, implicating the central canal of lower medulla. The entire floor of fourth ventricle, and the aqueduct of Sylvius, so far as the nucleus of the third nerve. (f) The degeneration of fibres of the cauda equina. (g) The involvement of the cerebral and cerebellar cortex and the inner capsule.

The general conditions to be drawn from the examination are, that contrary to the popular opinion, white and gray matter are equally attacked; that there is no point of predilection in the central nervous system for the appearance of the sclerotic patches; that the cortex of both cerebrum and cerebellum does not escape the degenerative process.



The presence of lesions in all cranial nerves is a matter of interest in view of previous opinion on that point. The degeneration of fibres of the cauda equina is of importance as throwing light on the possible extension of the process to the peripheral system. The sharply defined edges of many of the patches studied is worthy of mention, an observation contrary to Charcot's original description.

Secondary degenerations are not observed. Ganglion cells are in many cases entirely normal; in others, where the process is in a more advanced stage, varying conditions of degeneration, characterized especially by a much greater degree of pigmentation than normally occurs. Blood vessels, though in places somewhat affected, do not appear to stand in causal relation to the formation of the sclerosis, for the following reasons: (1) The patches are not always associated with diseased vessels. (2) The vessels, even in much degenerated areas, often show no marked changes. (3) Disease of vessels, in one case examined, was entirely lacking.

COLLINS.

**The Pathological Anatomy of Cerebro-Spinal Meningitis.** Dr. G. Bikeles (*Centralbl. f. Nervenkr. u. Psych.*, Aug., 1894) caused light epileptic seizures in guinea pigs by tapping them on the head with a percussion hammer (Westphal's method). Afterwards these animals showed symptoms of motor disturbances and muscular paresis, which in one case were accompanied by tonic and clonic contractions. Several weeks later the animals were killed. The brain and spinal cord were stained by Marchi's method (Muller's fluid and osmic acid), and examined microscopically. Throughout the entire central nervous system degeneration of the medullary fasciculi and sheaths were found. There were no hæmorrhages. Characteristic of all the cases was the fact that the degeneration did not limit itself to the seat of the injury. From these experiments it would appear that cerebral concussion causes a destruction of the medullary fasciculi in the entire central nervous system. The process may be considered as a traumatic degenerative neuritis, inasmuch as an injury may not only cause swelling of the axis cylinders but also destruction of the medullary sheaths.

MACALESTER.

**Changes in the Arteries at the Base of the Brain in the Sane and Insane.**—Mori was led to examine carefully the brains of seventy bodies, of which thirty-five died free from any mental trouble, and thirty-five were insane.

He found that of the sane thirteen presented anomalies of the cerebral arteries, nine of the cerebellar, and thirteen showed no variation.

Of the insane, thirty-two presented anomalies of the cerebral, one of the cerebellar arteries, and two were normal.

The anomalies in the cerebral arteries of the sane consisted in difference of calibre of the posterior communicans. In ten of the sane the posterior cerebral was given off from the internal carotid; in five the posterior communicans was absent; in nine the anterior communicans was duplicated; and in five there was found an accessory artery of the corpus callosum. — *Monitor Zoologico Italiana*, Anno 40, No. 10.

KRAUSS.

**Degeneration and its Stigmata.**—C. L. Dana, M. D. (*Med. Rec.*, Dec. 15, 1894). The final work of modern physiognomy will not be to fix upon human beings any stigma which marks them as necessarily useless, defective or dangerous. We have not discovered a type of criminal man, or of the insane man, or epileptic or neurotic man. All these have common marks, which show simply that they belong to the same somewhat handicapped family. Their presence in an individual will show that he must be especially careful in educating and using his natural power. The discovery of degeneracy throws an additional responsibility upon him, for there are few so bad but that, with a proper environment, they can get along successfully in life. There may be some born criminals, but they are very few; most are simply persons of

degenerate type who fail to husband properly the endowments they possess. We do not excuse the cripple who attempts to become a sprinter, nor should we excuse the morally defective who indulges in debasing habits and low temptations. All modern studies seem to show that man must be more than ever careful of his education, his training, and his surroundings, and of using all possible moral and spiritual agencies to strengthen his defects and make his powers more stable. The future of the degenerate depends enormously upon these factors, and his responsibility lies in his following that line of life which is right for him. By this means one can render many defects harmless, and make the unstable almost as firm in judgment and healthful in body as the happy possessors of perfectly balanced organizations.

FREEMAN.

**The Pathological Anatomy of Chorea Chronica Progressiva Hereditaria.**—Oppenheim and Hoppe (*Arch. f. Psych. u. Nervenkr.*, Bd. xxv., Hft. 3), observed two cases of this disease. One was a woman, fifty-six years old, who had been afflicted for sixteen years. The other was a man, seventy-five years old, who had suffered for five years. In both cases a hereditary predisposition was traceable. Autopsy showed the viscera to be normal. The brains presented, microscopically, considerable changes: narrowing of the convolutions in the motor regions, and of the parietal and occipital lobes; also, widening of the sulci. In the subcortical regions of these parts, small areas of fresh hæmorrhagic inflammation, and older ones with a fibrillary structure, were found on microscopic examination. There were also similar conditions in the pons and medulla. The pyramidal cells were normal, but the small round cells between the first and second cortical layer were diminished in number, especially in the motor region. In the cervical enlargement of the cord there was proliferation and swelling of the glia cells, and thickening of the meshes and blood vessels. The peripheral nerves showed distinct degeneration. The authors consider the process to be a disseminated cortical and subcortical encephalitis. The cortical atrophy is the consequence of the encephalitis. In the first case there developed hydrocephalus externus subsequently to the cortical atrophy. The changes in the cord are not to be considered secondary; they are caused by the conditions of the blood vessels, and are not systemic. The changes of the nerve cells are doubtful because the influence of *la grippe*, of which the first case died, and the old age of the second cannot be excluded.

MACALESTER.

**Histological Alterations in the Cerebral Cortex in Some Mental Disease.**—Colella (*Archives Italiennes de Biologie*, 1894, p. 216). Careful examination of the cortex of the brain in a case of progressive general paralysis with syphilis, in paralysis with alcoholism, and one of alcoholism, by the Gold method, has led the author to the following conclusions:

In general paralysis preceded by syphilitic infection, the histological changes are confined mainly to the blood vessels, to the neuroglia cells, as well as to the cell protoplasm and the protoplasmic branches. The axis cylinder prolongations are but rarely affected, and when this does occur it is rather late in the disease. Pathological alterations show themselves first in the blood vessels.

In dementia paralytica associated with or due to alcohol, there is found hypertrophy of the spider cells in conjunction with different stages of nutritional disturbances in the axis cylinder prolongations, with but rudimentary changes in the protoplasmic prolongations. In these cases the blood vessels do not show signs of degeneration.

In chronic alcoholism the changes are overwhelmingly parenchymatous in their manifestation. The axis cylinder prolongations are involved, and certain parts of the cell body and the protoplasmic ramifications. The blood vessels and neuroglia remain unaffected.

Finally, the writer concludes that the protoplasmic prolongations and the nerve prolongations have a different physiological significance, and especially have the protoplasmic prolongations a close relationship to neuroglia cells and blood vessels, so that they must play an important role in the nutrition of the nerve tissues. COLLINS.

***A New Theory of the Causes of Some Nervous Diseases, Especially of Neuritis and Tabes.***—Edinger.

The theory is based upon the following laws :

1. The functioning of every organ always causes molecular changes to take place in it; the organ suffers a certain damage which must be repaired. When this restitution is adequate to the amount of function, the organ is strengthened by functioning; in the other case it undergoes retrogressive changes.

2. A damaged or weakened tissue soon decays and makes place for the surrounding tissues which grow into it. What has heretofore been called proliferation, hypertrophy, interstitial inflammation, etc., has been proven by Weigert to be only an ingrowth of healthy tissue into diseased tissues.

In the so-called gray atrophies of the central nervous system the cardinal tissue is always first diseased, after which this weakened tissue becomes proliferated and retrograde; then destroyed by the neuroglia; This overgrowth of the neighboring tissues cannot but damage the whole organ. This overgrowth of interstitial tissue, caused by decay of the motor fibres in a nerve, must also damage the sensory fibres contained in it.

Edinger thinks that by appreciating the importance of these facts, many observations, heretofore unexplained, can be comprehended.

If there is a disproportion between function and restitution in such a manner that the restitution is not sufficient to cause the tissue to return to its former condition, a degeneration of the tissue must follow. This disproportion will occur in case of absolute overfunction when, for instance, a certain group of muscles or nerves is over-exerted. In some cases, even where the restitution would be complete for a normal amount of function, it would occur still easier if, aside from overfunction, restitution was impaired by deficient nutrition (as Edinger finds it to be the case in the so-called occupation paralysis of cigar rollers, etc.). But even with a normal amount of function nutrition may be so deficient as to prevent sufficient restitution. In the latter case those parts which perform comparatively the largest amount of function would have the greatest disposition to degenerate.

That there are diseases of the spinal cord which are intimately connected with disturbances of nutrition in the central organ, is proven by the existence of tabic symptoms in diabetes, by degenerations of the posterior columns or pyramidal tracts of dogs in whom artificial circulatory disturbances are produced by rapid rotation, etc.

It will also be easier to comprehend why multiple neuritis is seen accompanying or following such a large number of diseases (anæmia, tuberculosis, contagious disease, as measles, scarlet fever, etc.), by assuming that as a consequence of exhaustion constructive metabolism in most of these cases cannot furnish the amount of restitution which the (normal) function of nerve and cell requires.

Applying the theory to tabes, disturbances of equilibrium and ataxy of gait are the earliest symptoms of this disease, as those nervous apparatus which contribute the muscular sensations necessary for the maintenance of equilibrium and gait are used in an uncommonly high measure. The said symptoms, indeed, are the earliest and most important ones. We also find tabes chiefly in persons who over exert their legs (military officers, railroad employees, foresters, etc.), while we find it seldom in women who have more sedentary habits. This also explains the rare occurrence of tabes in puerle publicæ, although they are so fre-

quently affected with syphilis, the indirect etiological importance of which Edinger does not underrate.

The frequency of certain accompanying symptoms, as pupillary disturbances, atrophy of the tongue, of the peroneal muscles, muscles of larynx, etc., is also explained by the unusual amount of function performed by these parts. It is not strange, either, that ophthalmoplegia progressiva is frequently combined with degenerations in the posterior columns.

Edinger considers that the progressive character of progressive general paralysis is also satisfactorily explained by his theory. He is convinced that the nerve tissue is always first affected in this disease. The more nervous substance that is destroyed the more do the remaining healthy parts of it become exerted, which again causes further decay of nervous tissue, etc.

The numerous forcible arguments put forth in defence of Edinger's theory speak highly for its validity. Its application to the comprehension of tabes in its manifold varieties and of the progressive character of general progressive paralysis, seems particularly convincing. ONUF.

#### CLINICAL.

**The Differential Diagnosis of Encephalasthenia.**—By Dr. Julius Althaus (*Deutsche Zeitschrift für Nervenheilkunde*, Vol. v., part 6).

The diagnosis of encephalasthenia, often a very easy one, leads sometimes to fatal mistakes. It is especially liable to be confounded with one of the following: Cerebral tumors and general paresis in early stages of somewhat latent or irregular course; thrombotic softening of the brain, hysteria, hypochondriasis, and uric acid diathesis (lithaemia).

The absence of constant objective symptoms in encephalasthenia is principally responsible for the difficulty of diagnosis we are often confronted with in dealing with cases of this kind. According to the theory of the author that there are anatomically separated centres in the medulla for different vegetative functions, as, for instance, centre for the heart action, for the action of the glands, for the action of the kidneys, etc., isolated or combined functional disturbances are liable to occur in each of them, and produce clinical symptoms. Some of the latter show so much frequency and constancy as to justify their diagnostic importance, and prove of great value in comparison with the general limitation of objective symptoms in encephalasthenia.

Low specific gravity of the urine (1004-1002), neutral or alkaline reaction, and excess of phosphates are often met met with in encephalasthenics. Polyuria, slight degree of glycosuria (over 0.50-100) and constant or intermittent albuminuria are not very rare phenomena. Oxaluria is very rare, and by no means the cause of the encephalasthenic symptoms, as Prout believes. The careful examination of the urine in every "nervous" patient will help to avoid mistakes of diagnosis. Antipyrin has proved to be of nearly specific therapeutic power in the treatment of nervous (encephalasthenic) glycosuria.

Exaggeration of the tendon reflexes, especially the patellar reflexes, without muscular rigidity, though not constant, is a common occurrence in encephalasthenics.

Lastly, the absence of symptoms known to be produced by an anatomical lesion of the central or peripheral nervous system is in most of the cases conclusive.

The subjective symptoms of encephalasthenia are familiar; from the standpoint of a differential diagnosis the various morbid fears and paresthesias of the head will sometimes be of great help.

Cerebral hæmorrhage and embolic softening of the brain seldom need be taken into consideration in the diagnostic raisonnement.

Thrombotic softening of the Rolandic cortex, leading to general and local symptoms, gives rise sometimes to doubt. The age of the patient, encephalasthenia occurring in younger, softening in senile persons, will be of some aid. The occasional appearance of attacks of aphasia, monoplegia, etc., will decide the diagnosis. Subcortical softening has too characteristic clinical features to need further discussion.

Cases of cerebral tumors showing any of the known characteristic symptoms are easily understood in their clinical appearance. Some cases of latent or irregular course are doubtful for a while. The close study of the character of the head symptoms will often prove to be useful. The headache in tumor cases is constant, real pain, uninfluenced by drugs, and in contrast to the inconstant, proteus-like paræsthesias of encephalasthenies.

General paresis in the final stage and with more pronounced psychical symptoms requires often great diagnostic skill. Excepting the somatic symptoms in general paresis, much of the diagnostic aid is derived from the frame of mind of the patient. The euphoria of the paralytic contrasts distinctly with the depression of the encephalasthenie. Too much importance should not be attributed to a slight difference of the pupils or slight sluggishness in their reactions, since Magnus has shown that anisocoria is not so uncommon a phenomena in persons without any serious lesion of the nervous system. Moreover, the author believes that a functional lesion in the ciliospinal centre can give rise to various pupillary symptoms, as hippus, inconstancy, sluggishness, or inequality. Disturbances of speech and writing occur in paretics as well as in encephalasthenics. In the latter, though, the symptoms are not due to any lesion of the speech centre, but simply the cause of the impaired memory and temporary absence of mind. Polyphasia, polygraphia (*l'homme aux petits papiers*—Charcot), and the use of the exalted language are known to be encephalasthenic symptoms. Cases of so-called pseudo-paresis are, as to the diagnosis, often left in suspense. Hysteria, being a psychosis, is quite distinctly differentiated from the neurosis in question.

Hysteria affects more the lower psychical functions : feeling, disposition and instincts ; encephalasthenia the higher ones : perception, rationalization and volition. Distinct motor and sensory symptoms speak in favor of hysteria.

If nosophobia be regarded as not belonging to hypochondriasis, the diagnosis of the latter will rarely meet with great difficulty, showing more constancy and regularity in its symptoms. Nosophobia being always caused by external influences, epidemics, etc., and being so easily dispelled by the assertions of the physician, is in marked contrast to the imaginations and organ-hallucinations of the hypochondriacs. These are permanent, slightly, if any, influenced by the physician, and originating in the patient himself, without any detectable external cause.

Lithemia shows often just the opposite symptoms of encephalasthenia : pulse of high tension and high specific gravity of the urine.

FRAENKEL.

**A Case of Syringomyelia Associated With Hysteria.**—*Revista Sperimentale di Frenatria e di Medicina Negale*, Vol. xx., Fas. i., 1894. Dr. Cesare Agostini, of Perugia, describes a case of syringomyelia in a hysterical woman, thirty-five years old. Her family history reveals alcoholism, plithisis and nervous derangements. Her present illness began four years ago, with neuralgic pains in the left scapular region, and with fornication and a feeling of weight and debility in the arms, first affecting the pectoral region, then the arms, forearms, and lastly the hands. At the same time she developed hysteria, paraplegia and aphasia, and later on the classical convulsions of hysteria major. These symptoms persisted for two years and were finally treated successfully by hypnotism.

Muscular force of right hand thirty, of left five. Marked atrophy of the muscles of the forearm, of the interossei, and of the thenar and hypothenar eminences.

Sensibility—tactile sensibility is generally preserved, save on the posterior part of the left upper arm where hyperthæsia is present. Sensation of heat and cold and of pain are abolished from the neck to the ninth intercostal space and in the left arm. Muscular sense is intact, likewise sense of taste, sight and hearing. Visual fields concentrically contracted. Pupils do not react. Exaggeration of the muscle and tendon reflexes. Reaction of degeneration in the muscles of the thenar and hypothenar eminences.

After reviewing the literature on syringomyelia, the author concludes his article as follows :

1. Syringomyelia may be associated with hysteria, and confusion may arise when the difference pertains to sensibility alone.

2. The presence of progressive muscular atrophy, accompanied by diminished electrical excitability, persistence of the muscular sense, point to a syringomyelic affection rather than an hysterical.

3. The best diagnostic criterion in this and other neuropathic affections is hypnosis, in which muscles with anatomical lesions do not pass in to the cataleptic state.

KRAUSS.

**Caries of the Spine Causing Compression of the Cord Relieved by Laminectomy.**—Noble Smith (*Brit. Med. Jour.*, Dec. 1, 1894). The patient, a woman thirty-one years old, developed, after injury to the spine by lifting, a progressing weakness of the lower extremities which was associated with pain and anæsthesia. The pain was in the region of the sacrum, neuralgic in character, and periodically very severe. It was greatly increased by attempts at walking. Anæsthesia was complete as high as the gluteal fold posteriorly, and in front extended on the right side as high as Poupart's ligament, on the left nearly to the breast. The buttocks were hypersensitive; on the inner surface of the thighs, as far down as the knees, there was a narrow area of hyperæsthesia. Laminectomy, by removal of the neural arches of the eighth and ninth dorsal vertebrae, exposed a spongy material beneath the arch of the eighth vertebra, which was producing some pressure on the cord. The removal of this was followed by gradual recovery, so that five months after the operation she is able to walk without aid of stick or crutch. The spasmodic jerking of the lower extremities has ceased. Reflex aches have disappeared, as has also a sense of coldness of the lower extremities, which before the operation had been annoying. [The disappearance or the persistency of the hypæsthesia and hyperæsthesia after the operation are not referred to by the author].

COLLINS.

**A Case of Friedreich's Disease With Necropsy.**—J. M. Clarke (*Brit. Med. Jour.*, Dec. 8, 1894) reports a case of Friedreich's disease, which was complicated by the growth of a cerebellar tumor. The tumor was of very rapid growth and gave rise to such marked symptoms that these were recognized as a fresh addition to those of Friedreich's disease. The patient, sixteen years old at the time of death, had been affected since he was four years old. Neurotic family history, but no direct inheritance of the disease. An elder brother is affected in the same way. The symptoms which the patient exhibited were those typical of the disease. The unusual feature was the preservation of knee-jerk, but no ankle clonus; no increase of the deep reflexes or spastic rigidity. The symptoms pointing to brain tumor did not come on until less than a month before his death. They were vomiting, headache, inability to stand or walk, some convulsive seizures, lethargy, intense optic neuritis in both eyes, disappearance of knee-jerks, coma, opisthotonic spasm preceding death. The autopsy revealed a round lobulated tumor springing from the outer surface of the right half of the cerebellum, laying by the side of the pons and upper part of the medulla. Pathological examina-

tion of the central nervous system showed that the morbid changes affected chiefly the posterior columns, next the lateral, with involvement especially of the margin of the cord, with thickening in the pia mater and in the walls of the vessels and, probably, with some general excess of neuroglia, and small size generally of the cord. The changes were diffuse and nowhere had produced complete degeneration, and varied much at different levels. The cord was involved throughout its entire extent. The direct cerebellar tract was healthy. COLLINS.

**The Diagnosis of Extra-Medullary Spinal Cord Tumor.**—Pfeiffer (*Deutsche Zeitschr. f. Nervenheilk.*, Vol. v., 1894, p. 63). Male, 48, strong suspicion of syphilis in 1863. Probable secondary manifestations till 1871, but no apparent results of syphilis on examination. During past five years severe pain in the course of left sixth rib. No benefit from injections. Sensitive over sixth and seventh dorsal vertebrae. Exaggerated reflexes of lower extremities, especially marked on right side. Drags the left foot when he walks. Sphincters normal. Pain along sixth rib very severe and not controlled by narcotics. Girdle sensation, particularly on the sides. Lancinating pains radiating through legs. Motility and sensibility of trunk and upper extremities intact. No ataxia or Romberg's symptom. Diagnosis—Tumor of spinal cord (?) based principally on the severe, definitely localized, unattributable, uncontrollable pain. To relieve the pain, an operation consisting of section of the fifth, sixth and seventh extra-medullary dorsal roots, and extirpation of the thickened ganglion of the sixth nerve, was done. This was followed by important diminution of the pain; gradual appearance of interrupted conduction through the dorsal portion of the cord, till total paralysis of lower extremities resulted; anaesthesia from the umbilicus down, and paralysis of bladder and rectum. Cystitis and bed sores preceded death, which occurred two months after the operation. Autopsy revealed softening of cord in dorsal region, cystitis, pyelitis, and tumor of spleen. Histological findings—Myelitis of dorsal portion of cord, with ascending and descending degeneration and slight inflammatory thickening of both sixth dorsal spinal ganglia. COLLINS.

#### THERAPEUTICAL.

**Duboisine in Morphinism.**—Bernabée reports, in the *Bulletin della Società Lancis, and degli Ospedali di Roma*, 1893, a case of morphinism in a man affected with a neurosis of some months' standing, who injected subcutaneously 20 to 30 centigrammes of morphia daily. He presented all the phenomena of chronic morphine poisoning. All remedies, including hypnotism, were tried, but to no effect. Duboisine injected daily one-quarter to one-half millogramme, succeeded in a short time in curing the craving for morphine. KRAUSS.

**Subcutaneous Injections of Sulphate of Duboisine in Mental Medicine.**—By J. Massant (*Bul. de la Soc. de Med. Mental de Belgique*, December, 1893). In mental excitability of the insane the author considers the sulphate of duboisine in doses of from 1-100 to 1-75 gr., used hypodermically twice daily, a valuable medicament. The patient does not become accustomed to its use, and in addition to the mental quietude that it causes it induces sound and refreshing sleep. It occasionally, when given in larger doses, produces disagreeable but not dangerous symptoms, such as vertigo, nausea, vomiting, syncope, tachycardia, and, rarely, loss of consciousness, with or without twitchings of the muscles and incontinence of sphincters. When given in moderate doses, and its effects carefully watched, none of these symptoms result. COLLINS.

**The Treatment of Myxedema.**—Dr. John Julius Schmidt (*Deutsche Med. Wochenschr.*, Oct. 18, 1894) concludes that no permanent cure can be effected by the thyroid treatment. The administration of

the thyroid only takes the place of the normal thyroid gland for the time being. Schmidt recommends giving very small doses of the thyroid in substance at the beginning of the treatment, and gradually increasing. He begins with 1.0 daily, which method is less dangerous than the administration of large doses weekly. The same rule is also to be observed in the treatment with other thyroid preparations.

MACALESTER.

**The Electric Brush.**—(*Deutsche Med. Wochenschr.*, October 4, 1894.) The galvanic excels the faradic brush in the intensity of the cutaneous irritation, which can be localized and regulated quite accurately, and can be used as often as desired without causing any injurious results. Cutaneous irritation is useful in three different ways in the treatment of disease: First. It serves to stimulate the sensory and vaso-motor apparatus of the skin. The symptoms to be treated are peripheral anæsthesia, paræsthesia and circulatory disturbances (poor nutrition). The two former conditions are treated by applying the dry metallic brush, which is connected with the kathode, to the affected parts. It remains there until painful sensations are produced, is then taken off and applied again in a few seconds. In the treatment of *impotence* a small, moist metallic plate, that is connected with the kathode, is applied to different parts of the penis, and irritation is produced by opening the (strong) current frequently. Then a moist metallic brush, that is connected with the kathode of a weaker current, is used to stroke the penis, and is thus applied to the glans until a painful feeling is experienced. This procedure lasts three minutes, and is repeated every other day. In the course of three weeks carbonic acid baths are added to the cure, which alternate with the electrical treatment. Second. Cutaneous irritation serves as counter-irritation, and deeper parts, such as muscles and nerves which are in a condition of irritation, are often cured by this method. In the treatment of *sciatica* the patient lies on the belly; the skin is stroked over the whole length of the sciatic with a moist brush, which is connected with the kathode of a strong current, for five minutes, and the brush is allowed to rest repeatedly for a short time on the painful points along the course of the sciatic. This process is repeated daily, or every other day. Third. Cutaneous irritation serves as a means of acting upon remote parts of the central nervous system. In *tabes dorsalis* the galvanic brush exercises a most favorable influence on the bladder trouble. The skin over the lumbar region of the cord is stroked with a moist brush (strong current) daily until it becomes intensely red. The effect often begins after two or three applications, and in the course of a week the bladder disturbance is generally cured. The treatment should be continued for two or three weeks. The beneficial result lasts for weeks, months, and even years. The disturbances of cutaneous sensibility are also treated successfully by the galvanic brush, which is applied over the lumbar region, and then each lower extremity stroked for three or four minutes. One can observe that an extremity that at first does not feel the current at all, feels it painfully after a few applications. The affected parts regain their electro-cutaneous sensibility. The same principles hold good in regard to the sense of touch and pain. The subjective improvement of the patients is considerable; the distressing sensations of heaviness, cold, numbness and pain cease to a great extent. The gait also improves. Objectively a diminution of the ataxy is noticeable; the patients sway much less when standing with the eyes closed. The improvement is brought about by acting upon the nerve fibres that are not already degenerated.

MACALESTER.

**The Symptom Complex of So-Called Spastic Spinal Paralysis as a Partial Manifestation of a Hereditary Syphilitic Affection of the Central Nervous System.**—Hoffman (*Neurolog. Centralbl.*, July 1, 1894.)



A fourteen-year-old youth had, during the first months of life, manifestations of hereditary syphilis. During his first years he was physically and psychologically like other children. During school years he manifested intellectual and somatic backwardness. Although there was no diminution in possessed intellectuality, there was also no further development. During these years he did not develop physically as children do, yet he remained healthy and strong. In the twelfth year symptoms of bodily illness, such as stiffness and paresthesia of legs and hindrance of locomotion, appeared. One and a half years later disturbance of vision, probably due to paresis of accommodation, and six months after this he presented mydriasis, loss of pupillary response to accommodation and light, paresis of accommodation, spastic gait with exaggerated reflexes, but still preservation of strength in the stiff extremities, undisturbed sensibility, undisturbed sphincters and the bodily and mental condition alluded to.

The writer believes that this symptom complex points conclusively to hereditary syphilis, and not to some intercurrent condition. The psychical and pathological condition of character he believes to be due to lesion of the cerebrum, more particularly the cortex of the frontal lobes; the pupillary condition, analogous to that of tabes, and dependent on a similar condition. The spastic condition of the extremities is explainable by lesion of the white substance of the cord, especially the pyramidal tracks, which may have been primary or secondary to the changes in the brain. He believes the origin of the changes to be explainable by, first, a limitation of development of certain areas in the cerebral nervous system; and, second, by an active disease process of such areas. The reasons for not considering this case one of genuine syphilitic disease (syphilitic meningo-encephalitis and meningomyelitis), juvenile dementia paralytica—Little's disease—are briefly entered into. In conclusion, the author says the case may be considered a parallel to the cases of tabes so rarely seen in children. COLLINS.

**A Case of Alexia With Right-Sided Homonymous Hemianopsia (Subcortical Alexia-Wernicke).**—L. Bruns, M. D. (*Neurolog. Centralbl.*, 1894, Heft 1 and 2.) The patient was a woman, thirty-two years old. The disease began two years before death with continuous headache; afterwards vomiting, then visual disturbances and vertigo. Five months before death the diagnosis of a cerebral tumor was made. At that time the patient presented: Choked disc in both eyes, typical right-sided hemianopsia, associated with short attacks of complete blindness. Slight hemiparesis of the right side with patellar and ankle clonus. No disturbances of sensation, no pain or temperature. The patient further presented the symptoms of Wernicke's subcortical alexia or Freud's "optic aphasia."

Missing of substantives, especially of names for concrete objects in spontaneous speech, which otherwise was normal, aside from a slight paraphasia concerning mostly only single letters or syllables, seldom whole words. Repetition normal.

Comprehension of speech and musical understanding, unimpaired. Objects *shown* are recognized, but can seldom be named, although the first letter of the object's name is not infrequently found. Only exceptionally does the use of the other senses (touch, smell) enable the patient to *name* a concrete object. Printed and written letters are recognized, but cannot be named, even when the hand of the patient while writing is guided by the hand of the author. No words, except very short ones, can be recognized or read aloud (verbal alexia). Single ciphers are recognized and named directly or indirectly. Longer series of ciphers are not read. Spontaneous, and writing after dictation unimpaired at first, but gravely disturbed later. Copying impossible, but the examinations to prove this were somewhat defective. The autopsy showed the existence of three gliosarcomata of the left occipito-temporal lobe, which at

the time when the described symptoms of alexia were observed had not yet reached the cortex; at least a craniotomy performed one month before the patient's death, and exposure of the whole convexity of the occipital lobe and a large part of the adjoining parietal and temporal gyri did not reveal any tumor. The choked discs disappeared after the craniotomy, which, the author says, is a further proof of the theory that choked disc is not a true optic neuritis, but is the direct consequence of increased intracranial pressure. ONUF.

**A Case of Unilateral (Probably Congenital) Infantile Wasting of the Facial Muscles.**—M. Bernhardt. (*Neurologisches Centralbl.*, 1894, p. 2.) The patient, a man twenty-four years old, in whom a paralysis of the facial muscles of the right side was noticed two weeks after a natural birth. B. assumes that the paralysis probably was congenital.

The following muscles of the right side were atrophic, and did not respond to faradic or galvanic excitation: Frontalis, orbicularis, palpebrar, corrugator, zygomaticus, buccinator and the "nose-upper lip" muscles.

The right half of the orbicularis oris and the right depressor labii (quadratus menti) and depressor anguli oris muscles were somewhat flattened (with the exception of the orbicularis, which seemed to be even more voluminous than the left half), but otherwise distinctly marked; stronger currents were required to cause contractions of these muscles, but aside from this the reactions were normal.

No sensory disturbances, and especially was there no disturbance of the sense of taste in the anterior two-thirds of the tongue. Ocular muscles normal. The author seems inclined to assume a disease of the seventh nucleus as the cause of the muscular atrophies, although the history contains the statement that the case is one of peripheral paralysis of the facial nerve. ONUF.

**The Diagnostic Importance of the Achilles Tendon Reflex.**—Dr. Th. Ziehen (*Deutsche Med. Wochenschr.*, Aug. 16 and 23, 1894), after having observed and studied an extremely large number of cases, concludes that the Achilles tendon reflex is as delicate a test, if not even more so, than the knee-jerk. In certain diseases of the nervous system, the absence of the Achilles tendon reflex on one or both sides is of especial importance. Its absence in cases of mental disease points with great probability, firstly, to dementia paralytica, or syphilis of the nervous system, and, secondly, to senile dementia, and especially to chronic alcoholism. The significance of this symptom is naturally only to be depended upon if peripheral complications, such as sciatic neuritis, etc., can be excluded. MACALESTER.

**Circular Neurasthenia.**—Dr. Paul Sollier (*Rev. de Méd.*, No. 12, 1893) describes a form of circular neurasthenia, the course of which is analogous to circular insanity. Generally individuals with a hereditary predisposition are attacked; the onset, between the 18th and 25th year of life, is very gradual. There are periods of excitement and depression, the duration and intensity of which are extremely variable, lasting from several days to months. The chronic course the disease takes terminates, in severe cases, in loss of energy with hypochondriacal and melancholic attacks, which alternate with periods of excitement, and finally a general weakening of the intellectual faculties takes place. Outside of congestion of the head there is no evidence of somatic troubles. The prognosis is much more unfavorable than in cases of common chronic neurasthenia. The treatment is quite inadequate *quoad sanationem*; even in the relief of the symptoms, especially during the periods of excitement, it is unreliable. MACALESTER.

**Differential Diagnosis Between Injury of the Cauda Equina and the Lumbar Enlargement.**—F. Schultze *Zeitschr. f. Neuroheilk.* 1894, Vol. V., p. 247.

The patient, male, twenty-one years old, three years after a fall on the hips of twenty-five feet, suffered with pains in the loins and hips, radiating to the legs, numb feeling extending to the knees, incontinence of urine and feces. After nine months was able to stand and walk with aid of crutches. Gait unsteady, with knees rotated outward and on tiptoes. From eight dorsal vertebra lower down, sensitive; second lumbar vertebra somewhat prominent. Marked atrophy of lower thigh muscles; left gastrocnemius, soleus, tibialis anticus and posticus entirely paralyzed. Right thigh, all the muscles paralyzed. Gluteal region, both sides atrophied. Severe involvement of the muscles supplied by the sciatic and superior and inferior gluteal while those supplied by the crural and obturator nerves remain normal. Fascicular contraction of the atrophic muscles, especially the gluteus maximus on both sides. Complete reaction of degeneration in the muscles supplied by the peroneal nerves with the exception of peroneus longus and extensor Communis digitorum. In the calf muscles partial degenerative reaction. Simple strong diminution of direct electrical irritability in the glutei. Loss of patellar reflex on both sides. When the patella was tapped on one side there followed an inward rotation of other leg. No ankle clonus or plantar reflex. Cremaster reflex weak, abdominal and bladder reflex normal.

Incontinence of urine except with strong effort. Weakness of detrusor vesicæ and sphincter ani; no priapism, power of ejaculation and erection preserved. Sensibility in both thighs almost completely lost except in region supplied by saphenous. Marked anesthesia on posterior surface of thighs as far up as the middle of the gluteal region and of scrotum and perineal region. Just above and below Ponpart's ligament a zone of hyperæsthesia of the nerves, showing paralysis all come from the sciatic plexus except the posterior cutaneous of thigh and the external spermatic. The author asks, Are these conditions due to a primary injury of the cauda equina or to injury of the lower segments of the lumbar cord including the conus terminalis, or are both parts injured? That there was injury of the lower part of spinal column there is no doubt, but he inclines to the view that the spinal cord itself remained uninjured. He cannot entirely subscribe to Valentine's opinion, that the prognosis in injury to the cauda equina is favorable.

COLLINS.

**Rapid Cure of a Facial Paralysis**—Gustavo Lapez in *La Abeja Medica.*, Havana, An. iii., No. 4. A young lady, while going to a friend's house on a cold night, experienced suddenly a queer sensation, followed by a sharp pain in the whole left side of the face. The pain was most severe at the angle of the eye, and subsequently spread to the head. No improvement taking place, and becoming nervous and hysterical, she consulted the author, who found the left half of the face expressionless; the folds obliterated and the skin flaccid. The left eye was open—epiphora—and lagophthalmus; saliva not increased; uvula in its normal position; no disturbance of taste or of lingual sensibility; tongue deviates to the right, etc. The treatment consisted in vapor baths, followed by the use of the induction current. The paralysis occurred on the night of January 27, 1894, and after nine treatments the patient was discharged fully recovered February 16, 1894.

KRAUSS.

## Editorials.

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### THE BACTERIA OF MENINGITIS.

The rôle of the tubercle bacillus in the production of a very common and a very grave form of meningitis, now known as tuberculous meningitis, is sufficiently recognized. Are all the meningites equally microbic and equally infectious? Recent researches seem to indicate that they are, and that acute "idiopathic" or "simple" meningitis is as rare as acute idiopathic peritonitis *a frigore*. The latest foreign text books treat of meningitis as always infectious and secondary, and a new nomenclature seems to have arisen, or to be on the point of being adopted, in which we find described a *streptococcus meningitis* (very common), a *golden staphylococcus meningitis* (very rare), a *pneumococcus meningitis* (very common), a *typhoid* (Eberth) *bacillus meningitis* (following or accompanying typhoid fever), a *Klebs-Löffler bacillus-meningitis* (following diphtheria), and the list is likely to be indefinitely extended as the specific microbes of the grip, scarlet fever, small-pox, etc., become recognized and identified.

Guinon, in the *Traité de Médecine* (Charcot, Bouchard, Brissaud), of which the last volume is just published, treats the subject of meningitis with great developments from the standpoint of microbiology, and the same may be said of Dupré in the *Manuel de Médecine* (Debove and Achard), and of Ranzier in the last edition of Grasset's *Traité des Maladies du Systeme Nerveux*.

The labors of Netter, Fränke, Säger, Weichselbaum, etc., have contributed to establish the infectious nature of the meningites not tuberculous; they have found the streptococcus in the exudate of meningitis complicating puerpural septicæmia and erysipelas; and these same writers have reported cases of meningitis where the pneumococcus seemed to have been the infectious agent. These observations have been confirmed by Neumann, Ortmann, Runeberg, and others. The meningitis may occur in the course of pneumonia, or it may

occur independently of any pulmonary manifestations. Thus, out of thirty cases not preceded by pneumonia, Netter found sixteen that were clearly due to the pneumococcus. Netter was the first to affirm the pneumococcus nature of epidemic cerebro-spinal meningitis, and the bacteriological verification has been furnished in a certain number of cases by Leichtenstein, Fou, Bozdoni, Bonome and Uffreduzzi.

The most simple method of microbic infection of the meninges is that which results from a traumatism, which opens the way to germs from the exterior. Infection by *contiguity* is exemplified in meningitis by propagation from a phlegmon or an erysipelas of the scalp, furuncles and abscesses of the ear, caries of the petrous bone, mastoiditis, etc. The microbes in these cases of meningitis by propagation are generally the pyogenic staphylococci and streptococci (especially the omnipresent streptococcus.)

Interesting contributions to the literature of the infectious meningites have recently been made by Hutinél<sup>1</sup> and Grasset<sup>2</sup> in the *Semaine Médicale*. Both confine their observations to the pneumococcus form, or what Grasset, with a zeal for new terminology, calls *meningeal pneumococcy*. Pneumococcus meningitis may manifest itself in two different conditions. It may result from propagation to the meninges of an inflammation of the nares, ears, pharynx, organs in which the pneumococcus is a harmless inhabitant during health, or it may supervene as the result of a distant local lesion which has determined a general infection of the economy (acute lobar pneumonia). The meningitis not infrequently complicates an infectious (pneumococcus) endocarditis or pericarditis.

Hutinél has observed it in the course of suppurative arthritis of pneumococcus origin.

Pneumococcus meningitis is essentially a suppurative inflammation, and generally fatal, but some cases are on record which seem to point to the possibility of recovery from *mild* attacks (*frustes meningites suppurative*). Belfante and Auscher, as well as Hutinél, have recorded such cases. Dupré, in the *Manuel de Médecine*, also speaks of cases which he styles "false meningites," "meningism," in conformity with the "pseudo-peritonitis" of Bernutz, and the "peritonism" of Gubler. In these cases, we may either suppose a reflex irritation, causing

<sup>1</sup> *Semaine Médicale*, June 22, 1892.

<sup>2</sup> *Semaine Médicale*, March 7, 1894.

active fluxion stopping short of stasis and exudation, or a minor degree of disturbance—a congestion—caused by the presence of a microbe or its toxine, compatible with healthy resolution are complete *restitutio ad integrum*.

These views, according to which meningitis is always a bacterial disease, are somewhat exasperating to the old fogies of the profession who still contemplate with incredulity the progress and the conquests of microbiology, and who regularly every year at the medical meetings raise their voices and their protestations against the assumptions of bacterial pathology. The number of those, however, who are too hard headed and prejudiced to test for themselves by experimental work, and earnest, patient observation the claims of the bacteriologists, is becoming less and less every day, and this is owing to the patent fact that the results and methods of the latter are so open to the most critical observation and experimental control. If any one doubts whether in a given instance the pneumococcus is the pathogenic agent of meningitis, the microbiologist is able to point to a line of proof which certainly seems to satisfy all the canons of inductive logic. It seems to be well established, then, that in some instances, at least, meningitis is an infectious disease and due to a specific microbe. Is it so in all? It was formerly taught that alcohol is a prolific cause of inflammations of the brain membranes, but there really seems to be little proof that alcohol is ever anything more than a predisposing cause. The victim of alcoholism is predisposed to an invasion of the lungs, the endocardium, the meninges by the pneumococcus, or other infectious agent.

Neither gout nor rheumatism are any longer recognized as etiological in acute meningitis, and if syphilis may affect the membranes as a secondary(?) or tertiary manifestation, it may be said that syphilis has all the necessary qualities of an infectious disease, although the microbe is not yet known.

Instances are on record where meningitis has followed a contusion without any open wound, and such cases have been referred to in support of the claim that there may be a traumatic inflammation of the membranes in which microbes take no causative part. Perhaps this will long remain one of the principal arguments against the inclusion of all the meningites within the domain of microbiology. At the same time, the modern (germ

theory) pathologist has a ready answer to this objection; the traumatism caused the meningitis by playing the rôle of a provocative agent and making of the meninges a *locus minoris resistentiæ* for an infection originating in some other part of the body, or originating from without. The weakened meninges, here, afford what Jaccoud calls the "morbid opportunity."

It is evident that future studies and experimental researches under the most varied conditions are needed before the pathogeny of the meningites will be completely elucidated. We may, at least, be thankful to bacteriology for the light which this new science is shedding on these vexed questions of pathology.

#### AMERICAN NEUROLOGICAL ASSOCIATION.

The Council of the American Neurological Association has decided that the twenty-first annual meeting of the Association shall be held at Boston on June 5th, 6th and 7th, 1895.

There will be two sessions daily, one from 10 a.m. to 12.30, the other from 2 p.m. to 4.30 p.m.

It is hoped that every member will endeavor to make this meeting as successful as those in the past by contributing written communications, pathological specimens and microscopical specimens, or other matters of interest to the association.

The secretary calls your attention to Article IV. of the by-laws:

ART. IV.—The title of all papers to be read at any annual meeting must be forwarded to the corresponding secretary not later than three weeks before the first day of the session.

If it is desired to propose anyone for active membership, attention is called to Articles IV. and V. of the Constitution:

ART. IV. —Applications for active membership shall be made in writing by an active member at least two weeks before the first day of the session at which they are to be voted on. The secretary shall notify each active member of said nomination, provided, that by unani-

mous consent an application for membership may be made at one session of a meeting and acted on at the next session.

ART. V.—No one shall be eligible for active membership unless he has previously submitted a paper embodying original research, and which had not been previously published, on some subject connected with neurological science, which shall be referred to the council for examination and report.

The council desires to state that in all cases these laws will be interpreted literally.

The annual dinner will be held on Thursday evening, June 6th.

Very respectfully yours,

GREME M. HAMMOND, M.D.

*Secretary and Treasurer.*

## PRIZE OF THE AMERICAN NEUROLOGICAL ASSOCIATION.

The American Neurological Association offers a prize of \$200 for the best essay on any subject connected with neurological science.

The competition is open to physicians who are legal residents of States in North and South America.

Essays must be sent to the secretary of the association on or before the first day of May, 1895.

Each essay shall be accompanied by a sealed envelope containing the name and address of the author, and bearing on the outside a motto, which shall also be inscribed upon the essay.

Essays shall be type-written, in either the English or French languages, and with the pages securely fastened.

The council of the association reserves the right to reject any or all essays judged unworthy of the award.

Each essay must exhibit original research, and none will be accepted that has previously been published.

GREME M. HAMMOND, M.D.

*Secretary.*

58 West 45th Street, New York City.



THE REPORT OF THE STATE COMMISSION IN  
LUNACY OF THE STATE OF NEW YORK TO  
THE MAYOR OF NEW YORK IN REFERENCE  
TO THE INQUIRY INTO THE CONDUCT AND  
MANAGEMENT OF THE NEW YORK CITY  
ASYLUMS.

DATED DEC. 26, 1894.

This is a very important report and one which has been looked for with interest by a great many. To the New York *Herald* all credit is due for the forcing of an investigation into the management of the city asylums. Our State Committee of Lunacy had visited officially these institutions and was cognizant as far as any one outside the city asylums of the condition of affairs prior to the Mayor's request that a formal investigation be made. It is pertinent here to ask why they did not take up this matter before? But we must note here that the thanks of the profession and public are due to the New York *Herald* for being pertinacious and in successfully obtaining a verdict against the conduct and management of the city asylums from our "Commission." It looked very doubtful at first, but as the investigation proceeded very plain facts made it impossible to those who interested themselves in the matter not to see that a change far reaching and complete from one end to the other in the city's asylums management ought to be made.

The report excuses the physicians in charge. The justice or injustice in this is a question. The report criticises severely the conduct of all others in service, quality of food, accommodations, etc. Everything has been reviewed critically, except, as we have said, the medical officers. They stamp convincing disapproval upon everything else but them.

But space does not permit us to criticise at length this important report in detail, and we will only refer to the conclusion of the Commission :

"As before stated, the conclusion of the Commission regarding the evils which have been shown to exist in the New York city asylums is that they are largely, if not

wholly, attributable to the system under which these asylums are operated, and that, however feasible in theory, in practical operation this system has been a failure, and fallen far short of the hope which has, from time to time, been entertained for it. As a system it has developed inherent difficulties and defects, which experience has shown to be ineradicable, even under the ablest management, and which make its operation in all essential particulars practically impossible.

"Such being the case, the Commission would recommend that it be abolished, and that the policy of State care for the dependent insane of the city of New York be adopted at the earliest practicable date. It cannot be said that the present system of county care has not had a fair trial, and this, too, under exceptionally favorable conditions, as compared with any other instance of county care in the State, and yet it has failed to meet every reasonable or just expectation. If the system has been a failure from its inception, is it not reasonable to conclude that it is likely to be a failure for all time to come?

"Respecting the State hospitals, it is believed that most of them represent to-day all that is best in the present state of knowledge respecting the care and treatment of the insane, and that whatever other criticism may be passed upon them, it cannot be said that their inmates are not comfortably housed, properly fed, sufficiently clad, provided with sufficient attendance and given proper medical supervision and care. . . .

"The principle of State care for the dependent insane represents the most intelligent and humane thought upon the subject at the present time, and it is to be hoped that the local authorities in the counties of New York and Kings, who are responsible for the matter, will not long hesitate in taking the necessary steps for the complete consummation of the policy by availing themselves of the opportunity extended to them in the State Care Act."

We are not assured of our city's insane being taken better care of by the State.

The Commission, for instance, if they believe in their report, would allow the same medical officers to remain in power. No; this vast city ought to take care of its own paupers and insane. Separate the department of Charities from the department of Corrections, and both from politics.

Arrange it so the public benefactor and philanthropist can aid with good works and money, and make it

feasible that physicians of merit can enter into the active work as visiting and consulting boards. Is it not logical that New York City contains amongst its medical fraternity and its citizens enough material of brains, money and honesty to help these poor unfortunates?

Is it guaranteed that our State Board would do it better? Cannot our present Mayor aid us somewhat in this regard?

The last clause in the report will no doubt be interesting to many of our readers who are working to see our city insane asylums reorganized as hospitals, well equipped and doing scientific work. If we lose the city's control over these institutions there will be no hope for any chance of utilizing this vast material for study. As we know, the entire material of the State asylums is lost to advance investigation. The neurologists and alienists of New York City must try and do something in this matter quickly.

## Society Reports.

### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting held at the New York Academy of Medicine,  
Tuesday Evening, November 6, 1894.*

Dr. EDWARD D. FISHER, President, in the Chair.

#### PRESENTATION OF CASES.

Dr. JAMES J. PUTNAM, of Boston, briefly reported the histories of four cases of brain surgery recently coming under his observation, mainly in order to show the unexpected dangers attending operation. In all of these cases the chief symptoms were headache, nausea, vomiting and optic neuritis, and operation was advised for the purpose of relieving pressure. The first patient, a comparatively young man, did not recover from the effects of the ether, and died about five hours after the operation. No autopsy was permitted, but from an examination of the brain made through the opening in the skull evidences of a large hæmorrhage underneath the cortex were found. The dura had not been opened. The second case was that of a young girl who died during the ætherization, before an incision had been made. At the autopsy three tumors were found in the situation where the skull had been about to be opened. In the third case the patient's symptoms were somewhat relieved by the operation, which had been undertaken for relief of pressure. In the fourth case a tumor of the cortex was found, and a large cyst underneath the cortex. The latter was emptied, but it rapidly re-filled, and during the past few days there has been some oozing of brain substance from the wound and the patient is doing badly. These cases, the speaker said, go to show the dangers that attend opening the skull, which is usually regarded as a comparatively simple operation.

#### NOTE ON A CASE OF ACUTE POLIOMYELITIS IN A HEN.

BY DR. C. L. DANA.—The speaker stated that during the past summer, in the months of July and

August, there occurred in the neighborhood of Rutland, Vt., an epidemic of anterior poliomyelitis. Dr. Caverly, the Health Officer of the State, said that during this epidemic horses and fowls became affected, and on September 18 he sent Dr. Dana a large Plymouth Rock hen for bacteriological purposes. This fowl, on its arrival here, was paralyzed, and the owner of the flock, a physician, reported that several had died, the symptoms being similar to those noted in this one. The hen was taken to the Carnegie Laboratory and a careful clinical examination made, which revealed a paraplegia, not quite complete, and some paralysis of the wings; the head and neck muscles were not affected; there was no anæsthesia. Dr. Dunham, who conducted the bacteriological examination, inoculated several culture tubes from the spinal cord and meninges, and also took several sections from the cord and made smear stains, all with a negative result. The spinal canal of the animal was opened, and no evidence of meningitis was found, nor were there any signs of hæmorrhagic extravasation. On being placed in Muller's fluid and stained, a distinct area of softened tissue was seen in the central part of what might be termed the lumbar region of the cord. The destructive process was quite extensive, the congestion was intense, and there were several hæmorrhages. From the appearance of the cord it appeared that there had been an acute exudative inflammation, and that the process was so severe that necrosis came on before the inflammatory reaction; hence it is practically a case of acute infectious softening rather than myelitis. In conclusion, Dr. Dana said that while this case may not have any actual value, yet to a certain extent it corroborates the infectious theory of anterior poliomyelitis, and, so far as it goes, it supports the view that in this disease the changes are primarily vascular and not parenchymatous.

Dr. STARR stated that while on a visit to Vermont last summer he saw a number of the cases of anterior poliomyelitis referred to by Dr. Dana. Within a radius of perhaps twenty-five miles, about 160 cases of the disease occurred between the 25th of July and the 1st of September. Dr. Starr said he saw about a dozen of these cases. The epidemic was first regarded as one of cerebro-spinal meningitis, but from the lack of sensory symptoms and the peculiar distribution of the motor symptoms, the speaker said he regarded them as true

cases of anterior poliomyelitis. Sensory symptoms were present in some of the cases, but he has often found in anterior poliomyelitis in children over the age of twelve years that hyperæsthesia and stiffness of the muscles are among the first symptoms complained of.

Dr. B. SACHS said that in France the tendency has been to regard anterior poliomyelitis as epidemic. Epidemics of the disease have been observed in Stockholm. In his own practice he has noticed that by far the larger number occur during the warm weather. It is probable that we must include this affection among the acute infectious diseases, which is epidemic, or rather endemic. He has observed cases where it was difficult at first to say whether the disease was cerebro-spinal meningitis or anterior poliomyelitis. In several cases of the latter disease, in the very early stage, pain along definite nerve tracts was a very characteristic symptom.

Dr. A. JACOBI stated that last August he received a letter from Dr. Caverly regarding this epidemic in Vermont, in which he described a number of the cases, particularly emphasizing the fact that there was a good deal of hyperæsthesia. Basing his conclusion on this data, Dr. Jacobi said, he expressed the opinion that the epidemic was one of cerebro-spinal meningitis, as the manifestations of that disease differ widely at times. Since then, however, he has received more detailed information regarding the cases from Dr. Starr, who examined a number of the patients, and he now has no doubt that the epidemic was one of anterior poliomyelitis.

Dr. A. D. ROCKWELL stated that two of these cases were examined by him. In one the hyperæsthesia was very distinct, and he was inclined to regard them as cases of cerebro-spinal meningitis.

Dr. STARR called attention to the fact that in the classical descriptions of anterior poliomyelitis, so little mention is made of the pain and stiffness and hyperæsthesia. The reason for this probably is that so few cases of the disease are diagnosticated as such until they have gone on for a week or two, and by that time the sensory symptoms have practically disappeared. If this newer pathology of the disease is correct, and it is due to a congestion of the cord, sensory symptoms are undoubtedly present.

Dr. PUTNAM stated that at an autopsy made in a case of anterior poliomyelitis of two months' standing in an

adult, he found that the necrotic process was distinctly confined to the area immediately surrounding the vessels.

Dr. A. JACOBI said there are two distinct classes of cases in this disease. In one class, that referred to by Dr. Starr, there is fever and hyperæsthesia at the beginning, but these symptoms are readily obscured in cases where the myelitis comes on during the course of another disease, such as scarlet fever, pneumonia, etc. The acute symptoms are ascribed to the latter disease, upon recovery from which it is found that the child is paralyzed. In the other class of cases, and probably the large majority are of this character, the child is put to bed quite well, and when it is taken up in the morning it is found to be paralyzed; there is no fever and no hyperæsthesia; there is not even an outcry in the night.

Dr. PUTNAM said that in the case already referred to by him the hyperæsthesia was excessive; so much so, that he could hardly doubt that peripheral neuritis was also present. In all other respects the symptoms were typical of anterior poliomyelitis.

Dr. LANDON CARTER GRAY said he agreed with Dr. Jacobi that cases of anterior poliomyelitis like those last described are very commonly met with; yet there are a certain number where the patients have unquestionable sensory symptoms, which we are often apt to attribute to a neuritis. It is a fact that this disease usually occurs during the hotter months, and this subject was carefully studied some years ago by Drs. Weir Mitchell and Sinkler; they also showed that chorea is not dependent on temperature, but rather on barometric fluctuations.

Dr. G. M. HAMMOND inquired whether the diagnosis in the hen was based on the clinical symptoms or the pathological condition of the cord? In anterior poliomyelitis we are apt to associate a certain group of symptoms with certain pathological changes in the cord. In the microscopical sections exhibited by Dr. Dana the anterior horns appear to be symmetrical, and the changes do not exactly resemble those we find in the true disease.

Dr. DANA replied that in some of the sections, which he had been unable to bring with him, the lesion in the cord was very marked. In some there was a distinct loss of substance, with softening and hæmorrhage.

The PRESIDENT said that in the differential diagnosis between anterior poliomyelitis and cerebro-spinal men-

ingitis, the extreme muscular atrophy which we find in the former would not be so apt to be present in the latter. The electrical reactions, too, might aid us. Sensory symptoms, he thought, are often present in anterior poliomyelitis, and are apt to be overlooked.

### EXHIBITION OF MICROPHOTOGRAPHS OF NERVE CELLS (GOLGI'S STAINS.)

BY DR. M. ALLEN STARR.—These photographs were made by Dr. Edward Leaming, Instructor of Microphotography at the College of Physicians and Surgeons, from specimens prepared by Mr. Strong in the laboratory of the Biological Department at Columbia College. They showed various portions of nerve tissue, some stained by Golgi's method and others by a modification of that method employed by Mr. Strong. These microphotographs, Dr. Starr said, were exhibited this year at the British Association at Oxford, and received a good deal of favorable comment. One of them, showing the Purkinje ganglion-cell, took the prize in London for the best exhibit in microphotography.

### SOME OF THE IMPORTANT ASPECTS OF THE THERAPEUTICS OF THE NERVOUS SYSTEM.

Dr. JAMES J. PUTNAM read a paper upon this subject, the greater part of which was devoted to remarks on the importance of utilizing the newer psychological literature for the study and treatment of neurasthenia and hysteria.

A number of French and English writers, especially Janet and Myers, have made original researches of much value with regard to these subjects, but in America the investigations have excited comparatively little attention.

It is certain that the mental condition of neurasthenics must be understood if we would devise modes of treatment at once successful and satisfactory to ourselves, for the disease is largely one of mental origin.

The central idea would seem to be that the fixed ideas and morbid associations which create and keep alive many of the symptoms, even of neurasthenia, are usually not to be reached by influences brought to bear on the patient's reasoning and intelligence, because they have



their seat in regions of the mind which are ordinarily not illuminated by consciousness.

The curative influences on which we can rely are mainly of the following sort: the implantation of new ideas and interests derived from absorbing occupations of a useful character; the training of the patient's will, and, perhaps still more, the deep implantation of ideas of hopefulness and cheerfulness, the growth of which the patient may be taught systematically to promote; the inculcation of simple and robust systems of philosophy; and finally, hypnotism and kindred methods.

The experience of Van Eden, Schrenck-Notzing, and others have shown that neurasthenics can often, if necessary, be hypnotised; and though one may not wish to use this treatment himself, for one or another reason, yet all are bound to recognize what can be accomplished by it in skilled hands. The reader had seen abundant evidence of this in his own practice. The critical remarks of Forel and others show that the failures and dangers of hypnotism apply mainly to its improper or unskillful use. As adjuncts to direct mental treatment, it is often useful to follow out systematic and prolonged courses of treatment of other sorts, and among these static electricity is especially worthy of mention. It may or may not be of value in other ways, but it certainly is an aid to securing mental and muscular relaxation and making the patient more susceptible to auto-suggestions of useful kinds.

One objection to hypnotism and kindred methods of treatment is that the patient is liable to come into too close relations of dependence with the physician, but if this danger is recognized it may, in great measure, be avoided by a conscientious practitioner, and the relationship utilized for the patient's good.

Among American contributions to this general subject, the paper by Morton Prince on Association Neuroses, and that by Russell Sturgis on the treatment of fixed ideas by hypnotism, are especially worthy of notice.

Among other therapeutic measures referred to by the author, and which he has found valuable among dispensary patients, where the need of better methods for the treatment of nervous disease has long been felt, were gymnastics and massage, including the formation of classes for calisthenics. Treatment of this sort has been systematically carried out by professional masseurs (Dr. Lindström and Miss Colby), and by a number of pupils

from the Posse Gymnasium, all working without pay. The reader spoke of the great value of such an affiliation of hospitals and schools for gymnastics. During the past year he had treated fifteen cases of chronic spinal sclerosis by suspension, a method of treatment which he regards as wholly empirical and probably psychical. In almost every instance these patients declared themselves improved, both as regards eyesight and general condition. Frequently curious changes of temperature occur during the treatment. In several cases of tabes remarkably low temperature had been noted, independently of treatment.

In concluding his paper, Dr. Putnam stated that during the past few months he has tested the Flechsig treatment of epilepsy by means of large doses of opium, and he referred to a paper on this subject by Dr. Joseph Collins (*New York Medical Record*, Sept. 22, 1894). The treatment was employed in five cases; in none of them were the attacks wholly checked, but in all of them an improvement was noted. The opium was never pushed beyond ten grains daily.

Dr. STARR said it seemed to him that the reason why Dr. Weir Mitchell's rest treatment is so successful is that it combines most of the different elements which had been mentioned by the various speakers. There is first the expectation of taking a cure which has been of benefit to others; this is combined with a trip to Philadelphia, a change of scene and an entire change in the daily routine of life. The speaker also referred to the value of hydro-therapeutics, which makes an intense mental impression on the patient.

Dr. MARY PUTNAM JACOBI said that one writer, probably humorously inclined, has made the assertion that the beneficial effects of hydro-therapeutics are more pronounced in France than in England, because no French person ever takes a bath.

Dr. GRAY said that in some patients there is such an admixture of the symptoms of neurasthenia, hypochondriasis and hysteria that it is difficult to distinguish one condition from the other. There are, on the other hand, cases which we may term pure neurasthenia, and it is in these cases, as he understood the paper, that the author would employ hypnotism under the broad term of suggestion. Hypnotism, in the way that it is generally used, is an agent that we can seldom employ in neurasthenia, not only because there is a prejudice against it, but also

because it is difficult to impress such patients favorably by means of it. The same thing may be said about it in hypochondriasis, while on the other hand in hysteria its results are peculiarly favorable. As regards the value of electro-therapeutics, Dr. Gray said he is firmly convinced that the benefit following its employment in certain conditions is by no means confined to its influence on the mind of the patient. He has over and over again seen cases of neuritis in an advanced stage improve under the galvanic and faradic currents. He has seen marked benefit follow the use of this agent in many cases of anterior poliomyelitis and muscular atrophy.

Dr. WILLIAM H. THOMSON referred to the value of newness or change as a psychical therapeutic measure in nervous diseases, and narrated the histories of a number of cases in which remarkable improvement followed a change of scene and surroundings.

Dr. SACHS said it is claimed by some that hypnotism has not met with the reception in America which it deserves, and that one reason for this is that we are still subject to the old idea that the physician must prescribe; that we are altogether too fond of drugs. Personally, he regarded it as a therapeutic measure which is of value in comparatively few cases. After a thorough and impartial trial, his own experience with it has been anything but satisfactory.

Dr. MARY PUTNAM JACOBİ stated that she has found static electricity almost a specific for the purpose of dissipating muscular pains, whether rheumatic, hysterical or otherwise, even if such pain has entirely resisted the galvanic or faradic currents.

Dr. ROCKWELL said he has employed static electricity for many years, and expressed the opinion that its value has been very much over estimated; its slight quantity gives it very little value as compared with other forms of electricity.

Dr. JOSEPH COLLINS remarked that with a larger experience with Flechsig's plan he had not been led to change the convictions formulated when preparing his paper. He had never been enthusiastic about it, nor does he believe that it can in any way compare with the efficacy of bromides. In specially selected cases, however, particularly those associated with defects of development of the central nervous system in intractable cases and those in which physical aberrations are present, this plan of treatment will be found valuable. Opium is the most

important adjunct to bromides in the treatment of epilepsy. It has been his more recent experience that the large doses of opium originally advised are not necessary. If the daily quantity averages eight grains for the six weeks of its administration, the results will be quite as good as if fifteen grains daily had been given.

Dr. PUTNAM then closed the discussion.

At the close of the discussion a vote of thanks was given to Dr. Putnam by the Society for his interesting and able paper.

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## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, November 26, 1894.*

Dr. WHARTON SINKLER, President, in the Chair.

### EXHIBITION OF ANATOMICALLY CORRECT PLASTER CASTS OF BRAIN OF OURANG- OUTANG AND BRAIN OF CHIMPANZEE.

BY DR. JUDSON DALAND.

These casts were made by Steger, of Leipsic, under the personal supervision of Professor His, and are presented for inspection, in the hope that they may elicit some discussion with reference to general morphology and the distribution of the convolutions. In the model of the chimpanzee's brain the cerebellum is fairly well covered, but the posterior portion of the brain is much less convoluted than is seen in the model of the brain of the ourang-outang.

### EXHIBITION OF THE BRAINS OF THE CHIM- PANZEE AND OURANG BELONGING TO PROF. H. C. CHAPMAN.

BY A. P. BRUBAKER, M.D.

At the suggestion of Dr. J. M. Taylor, I have brought to the society this evening the brains of the chimpanzee and ourang for the purpose of affording the members

the opportunity of comparing them with the models presented by Dr. Daland. As to what extent they differ in their detailed structure can only be determined by careful examination. In the general arrangement of their leading fissures and convolutions they undoubtedly present many points of similarity. The one most marked difference between the model and the brain of the ourang is, the incomplete covering of the cerebellum by the cerebrum in the latter. This, however, probably depends on the age of the animal. The brain of the chimpanzee presents a less degree of conformity than its corresponding model.

## DISCUSSION.

Dr. F. X. DERGUM.—The chief interest in these brains lies not only in their relative simplicity as compared with the human brain, but also in the course pursued by certain fissures, especially the fissures of the occipital lobe. The brains shown to-night are evidently the brains of young animals. The brain of the adult chimpanzee is much larger.

The differences between the human and the anthropoid brains centre chiefly about the fissures and convolutions of the occipital lobe. These have already been described, and their significance much discussed.

Time would not permit our entering into an elaborate discussion of this interesting subject, and I will only speak of the peculiarity of the superior external bridging convolution—the *pli de passage supérieure externe*. In both the specimens and casts of the chimpanzee brains presented here to-night this convolution is represented as absent or submerged, *i. e.*, the external perpendicular fissure is freely confluent with the internal perpendicular. This would seem to justify the inference that this peculiarity was a constant feature, and could be relied upon as a distinguishing characteristic between the chimpanzee and human brain. However, at the meeting of the American Neurological Association held in Philadelphia, Dr. E. C. Spitzka exhibited the brain of a chimpanzee (Mr. Crowley, of Central Park fame), in which upon one side the superior external bridging convolution was fully developed up to the surface, *i. e.*, it presented the same morphological peculiarities as seen in man. On the other hemisphere it was absent (*i. e.*, submerged), just as in the specimens exhibited here this

evening. To make this point still more interesting, we must remember that in the human brain this convolution is every now and then (*i. e.*, exceptionally) found submerged.

Dr. CHARLES K. MILLS.—I am interested in these brains and in the brains of low-type human beings, which have many ape-like peculiarities. Fissural and gyral peculiarities have significance with reference to criminal lunatics and certain types of paranoiacs, and when we have made a sufficient number of observations on high-type and on low-type brains we will be able to say something more definite than this. These are teratological defects. I remember in the case of Beach, who killed his wife, the conditions presented in the occipital region were remarkable. The superior internal *pli de passage* convolution was unusually and peculiarly developed, and my own experience with the brains of criminals and of paranoiacs is that you not infrequently have submergence of the gyres, which are usually on the surface around the parieto-occipital fissure. The brain of Taylor, the man who killed his keeper at the Eastern Penitentiary, is the most ape-like human brain that I have ever seen, or of which I have ever seen a description. The fissures are unusually simple, and the occipito-parietal conditions much like those found in the chimpanzee. It is true that sometimes in high-type brains there is a simplicity of arrangement, as was the case in the brain from a very intelligent and highly cultivated person which was shown at a meeting of the American Neurological Society by Professor Wilder, but that fact in itself is not necessarily opposed to the view taken regarding the confluence of fissures and the submergence of convolutions. Many points must be studied in the clearing up of this subject.

The separation of the frontal lobe into four convolutions, which is looked upon by some recent writers as a high type peculiarity, was regarded by Benedict as peculiar to the criminal brain.

Dr. JAMES HENDRIE LLOYD.—I should like to ask Dr. Mills whether he thinks that chimpanzees and orang-outangs are necessarily paranoiacs? The fallacy involved is the assumption that a lower type necessarily means a vicious or abnormal type. It is taken for granted too readily that a slight reversion in type in the morphology of the brain convolutions implies a corresponding reversion in intellect and morals. The advocates of this doc-

trine must establish it on a much broader basis than they have yet done before it can be accepted as scientifically or legally valid.

Dr. CHARLES K. MILLS.—Whether the chimpanzee can be classed as a paranoiac I cannot say, but the chimpanzee is troubled very little with morals, and his tendency to theft and his exhibition of himself on all occasions, are such as to make Dr. Lloyd's argument of very little value. It is not a question of reversion to a low type, but a question of non-evolution.

Dr. F. X. DERCUM.—I agree in substance with what Dr. Mills has said, but I do not believe that single morphological peculiarities are of any great significance. It is the sum total of the conditions found in any one brain that should guide us. As a rule, the brains of murderers show an unusual number of these defects. What do these defects mean? They mean defective cortical development. Take the chimpanzee brain and lift up the operculum and you will find those convolutions which in the human brain are developed up to the surface. In the chimpanzee they are beneath the surface. On the other hand, there are some convolutions which are developed up to the surface in the ape brain, but which are beneath the surface in the human brain. This is the case with some of the convolutions on the inner surface of the occipital lobe; such, for instance, as the inferior internal bridging convolution. This condition, when it exists in the human subject (which is quite rare), is probably to be explained by such a relative retardation in the growth of surrounding convolutions as to leave structures, normally submerged, exposed on the general surface. Certainly, when such peculiarities occur in any number in a human brain, it is philosophical to regard it as a brain of low type.

Dr. JUDSON DALAND.—I am glad to hear this interesting discussion with reference to the morphology and arrangement of the convolutions. It seems to me that the study of morphology in relation to the brain, and also in relation to the internal viscera from this point of view, has been much neglected. Such study would be of distinct advantage, not only as regards the brain but also as regards the exact position and relation of the heart, lungs and abdominal viscera. In the method of making visceral models adopted by Steger, the body is frozen so that the organs are preserved, not only in their normal position and shape but also in their relation to other

organs. This gives an accurate knowledge that cannot be obtained from the ordinary dissections. For example, the topographical anatomy of the lungs and its relation with the heart are entirely changed after death from the entrance of air, causing marked changes in the shape and position of the thoracic and abdominal organs.

As water, when frozen, occupies more space than when it is a liquid, it is probable that this causes a slight increase in the size of all plaster of Paris models prepared in this manner.

Dr. CHARLES W. BURR presented

SPECIMENS FROM CASES OF PERNICIOUS  
ANÆMIA, SHOWING LESIONS OF THE  
SPINAL CORD.

DISCUSSION.

Dr. JAMES HENDRIE LLOYD.—In my observations on pernicious anæmia, I have found degeneration confined almost entirely to the posterior columns of the cord. I cannot say that my observations have been extensive, or that they are completed. I have not yet studied the sections thoroughly from the case that I have under observation, but the posterior sclerosis is so conspicuous that it can be easily seen. In some sections there appears to be a slight degeneration of the lateral tracts. I hope to have the opportunity to show these sections to the society.

Dr. CHARLES K. MILLS and Dr. J. W. McCONNELL read a paper entitled

LESION OF THE NAMING CENTRE, WITH  
REPORT OF A CASE INDICATING ITS LOCA-  
TION IN THE TEMPORAL LOBE. (See page 1).

DISCUSSION.

Dr. CHARLES K. MILLS.—It is not unlikely that this case may be criticised, for the separation of an area for this particular purpose, perhaps, has more opponents than advocates. This case offers strong evidence of the existence of such an area. This man was not word deaf.



The first temporal convolution is intact. The second temporal convolution was not intact. It had evidently been involved in the progress of the disease. The upper portion of the second temporal convolution and the fissure between the first and second temporal were not affected. The occipital region and the cuneus were not affected, nor was the anterior portion of the occipital lobe, except at the occipito-temporal junction. The hemianopsia, I think, was due to involvement of the optic radiations; it was not present when the verbal amnesia first appeared. We are warranted in saying, from an examination of the specimen, that this tumor began in the third temporal convolution and then extended in different directions.

Those who do not make a special study of localization sometimes get erroneous ideas of what we really believe when we speak of "idea," "concept," or "naming" centres, or centres of any kind for that matter. What we mean is, that here is a region to which converge the percepts from the different receptive centres of hearing, taste, sight, smell, touch, etc. Curiously this region is just the one anatomically so located as to receive with great facility impressions by way of association tracts which would come from the known receptive regions. A name arises in consciousness as a result of processes in this particular region. Ideation in a general sense is dependent upon the actions and inter-relations of the entire cortex. This is the region for concrete names. A patient like the one reported will use abstract nouns, like love and hate, and put them in sentences.

Dr. F. X. DERCUM.—I am one of those who have been loath to regard any special area as a naming centre. It seems reasonable, however, that the various impressions received from the eye and ear should at some point be combined; but if we pursue this thought to its legitimate consequences we must include the impressions from all of the senses. I admit, however, that the case reported is, clinically, very strong in favor of the view presented. It is not impossible nor improbable that there should be a centre in which impressions, especially from sight and hearing, are combined and symbolized. I infer that Dr. Mills regards it as a naming centre and not an ideation centre.

The PRESIDENT.—This case is of interest to me on account of a somewhat similar case which came under my observation several years ago. The patient, a lady

sixty-five years of age, had violent occipital pain, which was so severe as to require the free use of narcotics. With this there was complete left lateral hemianopsia. After two or three weeks, the pain subsided and she apparently returned to her normal health, the hemianopsia still remaining. I then, by accident, discovered that she had, to a great extent, lost the power of naming objects. This was three or four years ago. She has since regained to a certain extent the ability to name objects, and appears to be in good health, with the exception of the hemianopsia.

Dr. F. SAVARY PEARCE reported a case of

LOCOMOTOR ATAXIA, WITH SUDDEN ONSET  
AND UNUSUAL INITIAL SYMPTOMS; POS-  
TERIOR SCLEROSIS OF SIMILAR SYMPTOM-  
ATOLOGY IN PATIENT'S WIFE. (See page 8).

#### DISCUSSION.

Dr. CHARLES K. MILLS.—I cannot help thinking that the diagnosis of locomotor ataxia has not been made out. The point in favor of it is the absent knee jerk. It seems to me that the sudden occurrence of one of those peculiar lesions which sometimes develop in the region of the nuclei of the cranial nerves might explain it. Certain syphilitic lesions occur in the course of the root fibres, or at the nuclei of origin of these nerves, unassociated with true posterior sclerosis. This would not account for the lost knee-jerk. Knee-jerk is absent in disease of the cerebellum, and may be in some cases of quadrigeminal disease. This man has not the ordinary ataxic gait. He has an ataxic gait, but that it is the gait of posterior sclerosis is doubtful. Bruce and others have traced from the flocculus of the cerebellum fibres to the nuclei of the sixth nerve. A lesion of the cerebellum might account for it, or it might be accounted for in the same way by lesion of the tracts between the nuclei of these nerves and the cerebellum.

Dr. FRANCIS X. DERGUM.—Multiple specific lesions would explain the symptoms in this case. There may have been slight and previously existing ataxia, due to syphilitic infiltration of the posterior columns, and which remained unnoticed until the affection of vision directed

attention to it. Many ataxias do not notice the difficulty in walking until interference with vision occurs. It would be difficult to say how syphilis of the cord could be excluded in such a case as this. The occurrence in man and wife of a disease bearing such a close relation to syphilis in etiology is very suggestive.

Dr. M. V. BALL.—Might not the loss of knee-jerk have been present for some time? It sometimes happens that there is absence of knee jerk without any other symptom. This might be a coincidence in this case, for the man does not know anything with regard to his knee-jerk prior to the occurrence of the involvement of vision.

The PRESIDENT.—The knee-jerk is sometimes lost in cerebellar disease and in lesions of the corpora quadrigemina. A patient was under my care not long ago in whom quite suddenly inco-ordination of the right arm and leg occurred. There was no true loss of power, but there was great inco-ordination with a tendency to spasm. There was complete absence of the knee-jerk and considerable occipital pain and a tendency to stagger in walking, giving rise to the opinion that there probably was a cerebellar lesion. No syphilitic history was made out, but the man improved markedly under anti-syphilitic treatment.

Dr. F. X. DERCUM.—There are none of the other symptoms of cerebellar disease, no nystagmus, no vertigo, no vomiting, and a lesion of the cerebellum in such a position as to involve also the quadrigeminal bodies would be likely to present some of them; further, the absence of symptoms referable to the optic tracts also militates against this view.

Dr. PIERCE.—While there may be cerebellar disease in this case, it seems that the cord or its members *must* be involved from the fact that the numbness began in the perineum first, then in the left foot and leg up to the knee, and persisted there for a short time; then appeared in the right foot and left hand, and again, a few days later, in the right hand. This parasthesic condition has persisted from the first. These facts in connection with the sensation of walking on a carpet when stepping on boards, etc., would be in favor of the diagnosis of locomotor ataxia.

## EXHIBITION OF BRAIN TUMORS.

By F. X. DERCUM, M.D.

CASE I.—C. L., male, aged 18, student. Family and personal history negative up to one year ago. At that time he began to have epileptic seizures. They appeared to resemble ordinary attacks of petit mal, and recurred at intervals of three or four weeks until some three months before his death. The frequency then increased to two or three times a week. At this time, September 14, 1894, he was admitted to the Jefferson Hospital. Here the number of seizures were observed. While seated in a chair he would become suddenly unconscious, and would at the same time have slight general clonic movements. These were slightly accentuated upon the left side. The entire attack would last but a few minutes. Afterwards he would be slightly dazed. There was no local residual weakness, and the eye-grounds failed to reveal any change. He complained occasionally of headache, but this was never a marked feature of his case. In the intervals between the attacks he appeared mentally much depressed. He would sit quietly in his chair for hours without speaking, though he was not in the least degree demented. Two days before his death, which occurred on October 26, in a seizure, he wrote a long letter to one of his relatives. This letter did not differ from any that he had previously written. He had had an average education, and there was nothing in his letters to convey the idea that there was any marked loss of mental power. At the autopsy a large glioma, occupying the centrum ovale in the right frontal lobe, was found. It did not involve the cortex. It extended as far back as the anterior limb of the internal capsule. The growth presented traces of recent hemorrhage.

The practical interest of this case centres in the absence of optic neuritis and of the stupor and somnolence so often found in tumors of the frontal lobe. Probably the absence of these symptoms is to be explained by the fact that neither the cortex nor the membranes were involved, but that the tumor was situated well within the centrum ovale. The fact that headache was so little complained of finds a similar explanation.

CASE II.—E. A. B., male, aged 60, clerk. Family and personal history negative. No alcoholism or syphilis. Was well until September 15, 1893, when, on attempting

to sit down, he missed his chair and fell heavily to the floor. On attempting to rise, he noticed that the left arm and the left leg were slightly weak. He then went to bed, and slept well throughout the night. On the following day he resumed his work. He noticed, however, a little weakness and some awkwardness in his left leg. Four or five days later acute pain set in in the occipital region, extending thence over the entire head. It was very severe, and persisted uninterruptedly for two weeks. It then disappeared, and did not recur for two months. Examined January 12, 1894, it was found that he was slightly hemiplegic upon the left side, and further, that the movements on this side were decidedly awkward. No hemianæsthesia, no abnormality of speech, no involvement of the sphincters. Knee-jerk on the left side, pupils equal, and responsive to light; dynamometer, right hand 125, left hand 115. Examination of the urine revealed a specific gravity of 1042, and that it contained about ten per cent. of sugar.

Examined February 1, 1894, it was noted that an optic neuritis was present upon both sides, slightly more marked upon the right. The veins were greatly distended and tortuous. In addition, there was left lateral hemianopsia.

Examined on February 22, Drs. de Schweinitz and Thompson again confirmed the existence of left lateral hemianopsia and double optic neuritis. Many hæmorrhages were noted on the swollen discs. Pupillary reflexes to light and accommodation were normal. The pupils were equal in size. It was also noted that there was some diminution of the light reflex when a beam of light fell on the blind side of the retina, but there was no true hemiopic pupillary inaction.

He was now admitted to the hospital under the care of Dr. Weir Mitchell. Under dietetic treatment and codeine the amount of sugar became somewhat less. Headache was again more severe, however, and most marked during the evening. The patient seemed forgetful, and walked about as though somewhat dazed. From this time on, except as regards the diminution of the amount of sugar in the urine, there was but little change in his condition. Hemiparesis appeared to become gradually more and more marked, and in June it was noted that this side was also somewhat hemianæsthetic. On the 12th of June, while sitting up, he became suddenly faint, but did not lose consciousness.

After this attack it was noticed that the weakness of the left hand and arm had decidedly increased. The hemi-anæsthesia doubtfully present before was now more evident. He lost ground progressively from this time forward, becoming more and more demented and feeble until the 27th of July, when he died.

At the autopsy held the next day the right occipital lobe was found to be occupied by a rather firm mass, which in its more superficial portions resembled a sarcoma, while the more central portions were soft and jelly-like. Throughout its substance, and especially about the soft edges of the mass, which was in no sense circumscribed, but infiltrated the brain tissue, there were many small hæmorrhages, old and recent. Upon the lateral surface of the occipital lobe the tumor had perforated the dura at one point, and had slightly eroded the calvarium.

In contrast with the preceding case, this man presented marked optic neuritis and practically constant headache. The localizing symptoms, too, were such as to have led to a correct diagnosis of the seat of the growth during life. Anteriorly the tumor had invaded the centrum ovale above the internal capsule, and to some extent the capsule itself.

Adjourned.

## Book Reviews.

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DIE LEITUNGSBAHNEN IM GEHIRN UND RÜCKENMARK  
[*The Conducting Pathways in the Brain and Spinal Cord*].  
Von W. von Bechterew, mit 16 textabbildungen  
und einer lithographischen tafel. 210 pages. Leip-  
zig, published by Edward Besold. 1894.

The author of this book is known to neurologists as one of the most industrious investigators of the anatomy of the nervous system of the present day. During the past few years contributions from his laboratory and from his pen have appeared in various Russian and German journals, and this book is apparently the endeavor to place before the profession in a connected form these various contributions. The book is contained in five chapters. The first chapter is taken up with some remarks on the method of investigating the course of fibres in the central nervous system. Chapter II., the fibres of the spinal cord. Chapter III., the fibres of the crura cerebri. Chapter IV., the fibres of the cerebellum, and Chapter V., the fibres of the cerebral hemispheres. To the book is appended an exhaustive and valuable reference to the literature of the subject referred to in the text.

In discussing the methods that have been of service in revealing the secrets of construction of the nervous system, the author has given some attention to each one; he has laid particular stress, however, upon the embryological method, the one by which most of his results have been obtained. The method of comparative anatomy, the one by which Edinger has done so much valuable work, is somewhat neglected. We also miss special mention of the method employed by Marchi and Alzer; for although this can be grouped under the method of secondary degenerations, it has characteristics which justify its being placed in a category of its own. Although the embryological method is mostly favored, the author admits that for defining the longitudinal extent of systems of fibres in the spinal cord, it is less accurate and less satisfactory than the method of secondary degeneration; he ranks it first, however, for separating each single fasciculus from the surrounding parts.

Of the method of atrophy (Gudden's) he says that it is exact only so far as it gives definite results, but not when the results are negative; that is, if after destruction of a certain part, another part becomes atrophic, we can conclude with certainty these parts are connected with each other; but if for any reason the destruction of one part is not followed by atrophy in another part, which latter we have been led to believe from investigations by other methods is connected with the first, it would be wrong to conclude that the said two parts have any mutual relation. The author does not do Gudden's method justice by this general statement. It is necessary to explain more in detail by what principles Gudden's method is governed. Forel has clearly shown that the intensity of the atrophy depends upon the relation of the destroyed portion to the neuron, of which it forms a part. If it were an essential part of this neuron, for instance the chief axis cylinder of a cell of Golgi's first category, an intense atrophy of the ganglion cell from which the axis cylinder originated would follow. If, however, the destroyed part was but a collateral of an axis cylinder, or if it was the central part of a fibre which

terminates in a network in the near neighborhood, we would find only slight atrophy of the cell in the first case and usually only an atrophy of the said network in the second. As Forel explains in his last monograph (*Ueber das Verhältniss der experimentellen Atrophie und Degenerationen methode zur Anatomie und Histologie des Centralnervensystems*, Zurich, 1891), an accurate knowledge of the anatomical and physiological relations of the different parts can only be obtained by the use of all methods in such a way that the results acquired by one are completed, confirmed, and controlled by the others. This statement is also pertinent to findings by the embryological method. One cannot deny the great advantages of the latter, first, that it directly concerns the relations of the human nervous system, and second, that with less material and less work it gives a wider range of study than any other method, as it offers a mosaic-like differentiation not only of a single tract in different regions, but more or less in all the tracts in all parts of the nervous system simultaneously. Great as are these advantages the disadvantages must not be ignored. One of them is that the development of the myelin is rather irregular, so that one part of a nerve fibre may be medullated at a certain time, the other not; or one part may be surrounded with a thick medullary sheath while the myelin sheath of another part is still very thin, consequently the nerve fibre will appear coarsely club-shaped in one transverse section and finely club-shaped in another, while in a third section the conditions may be reversed again. The confusion liable to follow is evident. If it is a fact, as claimed by others, that hæmatoxylin stains not alone medullated but also non-medullated fibres, another disadvantage arises when hæmatoxylin staining is used, which Bechterew frequently does.

It cannot be denied that the embryological method can give us information as to the connection of individual fibres with ganglionic cells. Even with the comparative method of progressive serial sections this is sometimes possible, as, for instance, frequently the axis cylinder of a cell of the anterior roots can be seen passing, after covering itself with a myelin sheath, out as an anterior root fibre.

With the embryological method it is still much easier to follow the course of a nerve fibre, and prove its connection with a certain cell, since the confusion caused by the interlacing of fibres coming in various directions is lessened by the circumstance that part of them (medullated) differ in their appearance from the others (non-medullated). But such conditions are rather rare and those that have been cited are comparatively plain.

The method of atrophy and secondary degeneration have the great advantage over the others that they offer an isolation, not only of bundles or tracts of fibres, but also the cells with which they have connection. This isolation, it is true, is often negative, which is a disadvantage, because it makes it very difficult, for instance, to prove partial crossed connection with a certain group; a complete disappearance of a small number of cells of a certain group could only be stated when all the cells of the group are counted and their number compared with that of a similar group in a normal cord. In this case one would have to be sure that no section of a continuous series of sections was absent, and that the sections were of an even thickness; yet it must be said, when the methods of atrophy and secondary degeneration give distinct positive results, they are more reliable than results obtained by the embryologic method.

Bechterew states that the development of the various systems of fibres doubtless depends upon the development of the central apparatus in which they end, and for this reason the method allows us to draw other conclusions; such, for instance, as to the development of certain cerebral centres. This statement which, besides being too general, gives no exact idea in what it consists, and must be accepted with due reserve, particularly if it has reference also to spinal centres.



In the second chapter the author first describes the course of the fibres of the spinal nerves. Based upon the non-simultaneous development of the medullary sheaths two fasciculi of the posterior root are distinguished, the medial, consisting chiefly of coarse fibres, which, after entering the spinal cord, become partly portions of Burdach's and portions of Goll's columns, and the lateral, consisting almost exclusively of fine fibres which are more or less identical with Lissauer's column. The fibres of the lateral fasciculus have altogether different connections with the gray substance than the medial.

The fibres of the medial fasciculus take the following course :

A part becomes connected with Goll's column ; a part enters into the gray substance of the posterior horn, where they disappear at about the middle portion of the gray substance in the neighborhood of the cells situated there, and a part reaches the base of the anterior horns, where they disappear into a meshwork situated between the large cells of the anterior horns, while others still pass through the anterior commissure into the anterior horn of the opposite side.

The fibres of the lateral bundles of the posterior columns become connected with the substantia gelatinosa Rolandi, with the cells of the posterior horn generally, and especially with that group of cells situated nearest the substantia gelatinosa ; also with the cell groups situated between the lateral and posterior horns (lateral group of the posterior horn of Bechterew), and, lastly, through the posterior commissure with the gray matter of the other side.

B. discusses Sanger's and Munzer's conclusions on the course of the posterior roots, and admits that part of the fibres of Goll's column are a direct continuation of fibres of the posterior root, but concludes, from observations made by the embryological method, that part of them originate from the gray substance of the cord. He thinks that a direct connection of posterior root fibres (or of collaterals of these) with the nuclei *Gracilis et Cuneati* is not proven by Sanger's and Munzer's experiments.

In the outer roots he makes a systematic distinction between coarse and fine fibres, as the coarse fibres receive their medullary sheaths at a different period from the fine ones. The coarse fibres originate from the cells of the anterior horn of the same side and part of them also through the anterior commissure from analogous cells of the other side. The fine fibres take their origin from the cells of the lateral horn, from the *tractus intermedio-lateralis*, from the solitary cells of the posterior horn, and also from the cell group, which is situated at the anterior border of the substantia gelatinosa Rolandi.

Bechterew inclines to the view that the functions of the fine fibres of the anterior roots differ from those of the coarse fibres. He cites the theory of Gaskell and Mott, apparently with approbation, that the coarse fibres probably are destined for the innervation of striped muscular fibre, while the fine ones probably enter the sympathetic system, and are destined for the innervation of the internal organs.

The author classes the accessorius with the spinal nerve, and says that the cephalic portion is actually part of the pneumogastric nerve. He denies a connection of the accessorius with the nucleus ambiguus and with the solitary fasciculus of the medulla.

The following secondary connections of the various cell groups are assumed by B. :

First. For Clark's columns with the direct cerebellar bundles, with Burdach's and partly with Goll's columns, and through the anterior commissure with cells of the anterior horn of the other side.

Second. From the cells of the central group of the gray substance many fibres pass into the lateral columns, while others enter the anterior commissure.

Third. From the cells which are situated at the anterior border of

the substantia gelatinosa Rolandi, and which B. considers to be the most important terminal station of the fibres of the lateral fasciculus of the posterior roots, through the anterior and partly through the posterior to the lateral columns of the other side and to Burdach's and Goll's column.

He cites Ramon y Cajal's observation concerning the division of the posterior root fibre into ascending and descending fasciculi, the collaterals of these branches, etc. The ascending systems of fibres originate from the chief axis cylinders of cells of the gray substance (for instance the direct cerebellar fasciculus); those of the descending systems (first pyramid) end with terminal branches in close neighborhood to the cells of the white substance.

B.'s description of the coarse fibres of the white substance of the cord is chiefly based upon results obtained by the embryologic method and that of secondary degeneration.

First. Burdach's column. In the grouping of its fibres B. accepts Flechsig's division of three zones. He thinks that besides direct posterior root fibres Burdach's column contains a system of short fibres which connect different levels of the gray substance of the posterior horns.

Second. Goll's column. The results of the embryologic method justify the division of a median, lateral and intermediary zone. The median zone contains fibres which are the direct continuation of the fibres of the posterior root.

Third. The antero-lateral columns. He distinguishes six tracts. First, the lower pyramidal tracts; second, the direct cerebellar tracts; third, the medial fasciculus of the antero-lateral column; fourth, the antero-lateral or antero-external fasciculus (Gower's tract); fifth, the ground bundle of the antero-lateral column, and, sixth, the anterior pyramidal tract.

The median fasciculi of the antero lateral columns have been discovered by the embryologic method. It forms that part of the border of the gray substance which is situated next to the lateral border of the gray substance. Of the origin of that fasciculus nothing positive is known. In opposition to Lowenthal, Bechterew emphasizes that the direct cerebellar fibres originate from Clarke's column. They are connected with the cerebellum through the Restiform body.

The fibres of the antero lateral fasciculus can be traced into the anterior conducting paths of the medulla, where they probably become interrupted.

The author cites the interesting observations of Barcacci, that lesion of the cord at the level of the twelfth vertebra caused ascending degeneration of Gower's tract, and left the direct cerebellar fasciculi normal, but that lesion at the level of the sixth dorsal vertebra caused ascending degeneration of Gower's tract and of the direct cerebellar fasciculi. The area of the lateral pyramidal tracts, as shown by the embryologic method, shows much larger than when isolated by the method of secondary degeneration. B. contends, therefore, that the former must contain fibres of other systems besides those of the pyramids.

The third chapter, which is taken up with a description of the course of nerve fibres in the cerebral stems, begins with a description of the various nuclei and nests of gray matter situated in the peduncles.

He mentions a nucleus situated at the level of the nucleus of the lateral fillet in the central part of the formatio reticularis. This nucleus, which B. thinks has not been described before, he proposes to call nucleus centralis superior and centralis, and for purposes of distinction he proposes to call the superior central nucleus, nucleus centralis superior medialis. In speaking of the functions of the various nests of gray substance situated in the cerebral stems, he says that based upon

experiments performed by him he must describe equilibratory functions to certain parts that are situated near the third ventricle, and which have not yet been sufficiently investigated anatomically. The nuclei of the pons have relation to the maintenance of the equilibrium. According to Misslawsky's experiments the nucleus of the anterior column must be a respiratory centre. The lowest central nucleus corresponds closely to the location of the vaso-motor centre. This justifies the statement that the fasciculi of this centre is perforated by said nucleus. The nucleus reticularis, most probably, is a particular motor centre.

B. says that the superior olivary bodies being in connection with the nucleus of the acusticus (nucleus anterior Meynert's), we must ascribe to them the functions of a centre governing the reflectory movements of the eye. He does not doubt that the corpora bigemina posterior besides having auditory functions, have the significance of a special motor or coördinating centre. The nuclei of the optic thalamus have chiefly motor functions, governing especially the involuntary movements of internal organs, heart, stomach, etc., and those involuntary movements which are the expression of sensations or psychical reflex.

B. then describes the course of the fibres of the cerebral nerves. In opposition to Lauras, he denies the relation of the twelfth nerve to the nucleus ambiguus; also to the inferior olivary bodies. This in opposition to the opinion of Forel and of Mayser. He reiterates the assertion that the pneumogastric is connected with the nucleus ambiguus of both sides, although Mayser, and more recently Koch, denies this connection. The author has discovered a new nucleus situated near the bottom of the Calamus Scriptorius, latterly from the nucleus of the ninth nerve. He is sure that this newly discovered nucleus is also connected with the ninth nerve. B. still maintains that the anterior root of the eighth nerve is connected with the lateral or Deiter's nucleus, and says that investigations with Golgi's method by Sala, who denies these connections, are not reliable or convincing. Part of the fibres of the seventh nerve originate from the nucleus of the other side. He thinks that a connection of sixth nucleus with the third nucleus of the other side has yet to be proven. The cells of the substantia ferruginea probably have relations with the fifth nerve, according to pathological and anatomical investigations of Meitzel. B. is satisfied from observations by the embryologic method that the crossing of the fourth is complete and not partial. There is no doubt that part of the third root fibres are connected with the nucleus of the other side.

The course of the second nerve is then described at length. Of Gudden's commissure it is assumed that it probably serves to give a crossed connection of the corpora geniculata interna with the lenticular nuclei.

One portion of the fibres of the tractus opticus pass to the gray substance of the third ventricle. Of the paths of the optico pupillary reflexes, nothing positive can be concluded from B.'s description.

B. then mentions the results obtained with the silver method as regards the histologic connection of the cerebral nerves with their nuclei. The root fibres of the ninth and tenth nerves, according to Held's investigations, divide into ascending and descending branches analogous to the posterior spinal roots. The descending branches form the solitary fasciculi.

The anterior root of the eighth nerve also divides into ascending and descending branches; the last are none others than the ascending acoustic root. The first forms the remainder of the vestibular root which ends in Deiter's nucleus and the nucleus vestibularis and posterior nucleus. Similar branching takes place in the fifth nerve; the descending branch forms the so-called ascending root, the collaterals of which blend with terminal branching of the substantia gelatinosa.

In discussing the influence of the cerebellum on maintenance of

equilibrium, the author says that lesion in vicinity of the third ventricle, as well as in the vicinity of the inferior olive, produce entirely analogous disturbances of mobility to that produced by lesion of the semicircular canals or of the cerebellum, and further, that lesion of certain parts of the restiform bodies, the thalamus and section of the acusticus will cause loss of ability to maintain equilibrium. It is unquestionable that this statement by Bechterew tallies with clinical observations, particularly in cases of tumors, neoplasms and embolus of the mid brain, and is an important fact to keep in mind.

The fillet in the crura is divided into four portions: 1. The general fillet, which is the essential continuation of the fillet, and extends through the entire cerebral crura. 2. The lateral or inferior fillet, which lies laterally to the general fillet and reaches from the apex of the superior olive to the nucleus of the posterior corpora quadrigemina. 3. The scattered bundle thus passes forth from the base of the cerebral peduncles and penetrates nearly the entire breadth of the general fillet, especially the medial portion of the latter, that is, from the level of the substantia nigra to the under portion of the pons. 4. The medial accessory bundle of the fillet passes out from the base of the peduncles of the cerebrum and lies medial to the fillet.

The divisions of the fillet are to be recognized not alone by their position and development, but also, and especially by their period of development. The lateral or inferior fillet is the first to develop, about the sixth month of foetal life. About the full term of foetal life the main fillet is developed, shortly after birth the scattered portions of the fillet, and last of all, the accessory bundle of the fillet.

Serial sections of the brain and spinal cord made from a thirty to thirty-five c.m. long foetus, show that from the bundles of the funiculus cuneatus, the lateral circuit of the fillet take their course in two different directions. One set beginning at the height of the posterior corpora quadrigemina pass along the periphery of the crura cerebri, and bend posteriorly; shortly thereafter they take their course in company with the fibres of the lateral fillet found here to the corpora paraventricularia, in part, while a portion sinks into the anterior corpora quadrigemina. The other set take course farther forward; at the level of the upper portion of the red nucleus they begin gradually to bend laterally and repair to the nucleus of Luy. A portion of the fibres experience here an interruption; the greater portion, however, pass laterally and superiorly to Luy's nucleus on one side to the ansa lenticularis, and unites itself with the first and second portions of the globus pallidus, while the other side, through the mediation of Meynert's commissure, goes to the globus pallidus of the other side. Meynert's commissure serves also to give a crossed connection of the lenticular nucleus with Luy's body. The connection which was formerly supposed to exist between the corpora geniculata by means of Meynert's commissure has been entirely disproven by Darkschewitsch and Pribytkow.

It is impossible to give the pathway and connections of fillet as stated by the author in greater length, except to say that the lateral fillet is considered to be the most important tract for the sensation of hearing, inasmuch as the nucleus of the anterior corpora quadrigemina in which the inferior fillet is interrupted must serve as a station through which auditory excitation must pass on its way to the centre.

The pages devoted to the fillet are among the most lucid and convincing in the book, and no student of anatomy can afford to overlook them.

It is not only possible, but very probable, that the same centres in the cortex which are now associated topographically with certain functions may, through its many sided connections with the periphery of the body at one and the same time serve to perform several functions.

Certainly there have been cases observed where affection of the

cortex produced disturbances of sensibility. But in these cases one finds in an overwhelming majority of the cases the lesion either beyond the borders of the motorial area, or it involves not only motor areas, but other cortical areas. Examination of the literature of such cases leads Bechterew to the conclusion that the parietal convolutions are by far the most common seat of lesion when there is disturbance of skin and muscle sensibility.

There are found in literature cases in which lesion of the lower portion of posterior cerebral convolution was accompanied by disturbances of sensation. These observations B. thinks are in accord with his observations in animals, which experiments led him to the conviction that in animals which were subjected to a destruction of the posterior and outer part of the motorial fields (corresponding to the parietal convolution in man) there followed disturbances of skin and muscle sensation in the contra-lateral extremities, and at the same time evidences of paresis of the muscles, such as are observed after lesion of the motor areas did not present. Moreover, destruction of the posterior outer portion of the gyrus sigmoides (dogs, cats) and the posterior cerebral convolutions (monkeys) which areas includes motor centres, calls forth disturbances of sensibility.

We must, therefore, conclude that the motor and sensory centres are quite independent, but they lay very close to each other, and not infrequently in a certain area they lay one over the other. In reference to the corpus collosum, the author gives nothing new. The point of exit of one portion of the fibres of the balken corresponds in the one hemisphere to the origin of the other part in the other hemisphere. From the middle of the ventricle roof the fibres of the balken go toward both sides, asunder, in the form of a fan, and cross in the inner half of the hemisphere with ascending bundles of fibres of the crura cerebri, and reach therewith the cortex of the outer and upper part of the hemispheres. A greater part of the fibres which pass out from the anterior end of the balken take their way forward in the form of a bow to both anterior lobes (forceps anterior). The fibres of the posterior end of the corpus callosum describe a similar bow-like curve on their way to the posterior segment of the hemispheres (forceps posterior). The course of fibres of the rostrum of the corpus callosum ultimately tend to the basal surface of the anterior convolutions, which Henle wished to have known as the white commissure of the base.

The so-called tapetum has been shown by new investigations not to belong to the balken as was formerly assumed, but is a continuation of the inferior half of the balken's terminal, *fasciculus longitudinalis inferior*.

Embryologic methods corroborate the results of Monakow on the one hand and Vexas on the other, that Deiter's nucleus is in connection with the lateral tracks, especially the ground bundle of the cervical cord, and that it has no connection or relation with the anterior or posterior columns. Bechterew considers that the bundle described by Bruce as going from the inferior olive to Deiter's nucleus is but a part of the continuation between the anterior ground bundles and Deiter's nucleus which he formerly described.

With Kolliker and others, Bechterew believes that the sensory cerebral nerves spring from extra cerebral ganglia.

In reference to the ending of the optic nerve, the more recent investigations of Ramon y Cajal show that a considerable portion ends undoubtedly entirely free, with rich brush-like ramification, in the optic centre of the mid brain. Independent from this the optic nerve contains fibres which originate from the axis cylinder of cells in this centre. These fibres belong in their free endings to the retina. On the other hand, we find between the numerous terminal branches spindle-shaped cells imbedded in the mid brain, the protoplasmic branches of which everywhere come in contact with the terminal branches.

The extensive relations that the optic tract gets through contact with branches of nerve endings is stated in considerable detail.

The cells of the cerebral cortex have no continuous anatomical connection one with another, but functional relation between cells occur in the following ways: 1. Through contact of protoplasmic branches of adjoining pyramidal cells, as is the case in the multipolar cells of the anterior horns of the spinal cord. 2. Through contact of collaterals of cellular axis cylinder branches, which in greater or lesser numbers sink into a texture of fine nerve fibrillae. 3. Through contact of end branchings of nerve fibres and collaterals of axis cylinders with protoplasm end branchings.

The book closes with a few pages devoted to a grouping together in numbered paragraphs of the principal conducting paths in the central nervous system, and to those who are prevented from carefully perusing the entire book these paragraphs are commended. They will also be serviceable to him who has given careful study to the contents of the volume, impressing, as they do, the salient points. Sixteen diagrams illustrate points discussed in the text. These diagrams, although many of them are not entirely new, are ingeniously conceived and in most instances intelligently applied. Some of them, however, need modification; for instance, one which represents a cross section of the spinal cord. As it is now, it shows fibres from the posterior root ending in cells of the posterior horns, and at the same time these cells give out fibres which pass ventrally and then leave the cord in the shape of anterior roots. Such a double function or purpose was formerly attributed to cells but newer investigations have shown that such views are no longer tenable. We must confess to a disappointment that the author has not seen fit to give drawings illustrative of anatomical conditions, in the interpretation of which he is not in accord with other investigators.

We can imagine the value of the book enhanced and its usefulness more widespread if it contained such drawings. The colored plate does not call for special mention either to commend or to condemn.

In conclusion, we desire to recommend in no uncertain terms to every earnest student of neuro-anatomy this book. It is without question the most important contribution in its line that has yet emanated from the school of Flechsig. By its publication the author has fortified his reputation as an investigator and as an anatomist, and made neurologists his debtor.

JOSEPH COLLINS.

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TRAUMATISM AS A CAUSE OF LOCOMOTOR  
ATAXIA: A CRITICAL EXAMINATION OF  
THE EVIDENCE, WITH REPORTS OF THREE  
NEW ALLEGED CASES.<sup>1</sup>

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THE liability to litigation of cases of locomotor ataxia alleged to be due to injury gives to the matter of this paper considerable practical importance. Most of the text-books from which practitioners derive their opinions in such cases state that trauma is a cause of this disease. It is, therefore, important that the correctness of this opinion be either established beyond question, or, if this opinion is not well founded, that this fact be shown. On three occasions I have been asked to give an opinion as to whether the existing locomotor ataxia was, or was not, caused by an accident. Two of these cases were the subjects of litigation. I will mention, first, those main facts of the second case that are pertinent to this inquiry.

On the 29th (?) of March, 1890, a man stepped from an electric street car while it was in motion. The car had almost stopped, but just as the patient stepped off it started again. He somehow missed his footing and fell

<sup>1</sup> Paper presented at the meeting of the American Neurological Association, May, 1894.

forward, striking upon his face and chest. At the time, he felt "a curious feeling" (not a pain) in his back. It seems to have been the jerk of the car that caused him to miss his footing. There were no particular local injuries at the time, excepting a sprained wrist, but that evening he states that he had some nausea, and he lay in bed the next day, Sunday, to rest because he felt weak and faint. There do not seem to have been any special symptoms at the time, excepting a sprained wrist, which prevented him from working at his trade, that of a book-binder, although he continued to go regularly to the workshop where he superintended the work. At the end of one or two weeks the wrist was well.

In the following June, that is, between two and three months after the accident, for the first time he began to have tabetic pains in the feet and legs. For these he attended the Homœopathic dispensary, where the physician in charge pointed out to him that he had ataxia. He was not conscious of this later fact until his attention was called to it at this time. After this, he noticed that he staggered and had some dizziness. From this time on, tabetic symptoms seem to have developed with considerable rapidity, so that when I first saw him in the early months of 1894 (three and one-half years later), he presented an extreme picture of posterior sclerosis. A careful inquiry into his previous history, as well as an examination of his body, failed to disclose any evidence of syphilis.

It is worth mentioning that on August 3, 1890, that is, about four months after the accident just related, the patient met with a similar accident, falling in the same way as he was getting off a street car. Up to this time he was fairly well, but from the date of the second accident his condition rapidly grew worse. I gave the opinion that the spinal disease in this case was probably not due to the accident, but that an incipient tabes, with unsuspected ataxia, had existed previous to the injury, and that the patient had probably fallen in consequence of the ataxia.

My reasons for this opinion were:

First. The slight nature of the injury.

Second. The absence of any examination at the time of the accident, to show that the patient was free from this particular disease.

Third. The patient did not know he was ataxic until the fact was pointed out to him by a physician. After



this, he recognized that he staggered, and, in fact, had advanced inco-ordination.\*

It is evident, then, that ataxia must have existed for some indefinite time previous to his visit to the dispensary. How long a time? If a day, it may have existed a week, a month or two months (the time that had elapsed since the accident). We are all aware that tabes may exist for a long time before its presence is discovered. It may be that a patient, on being tested, is found to have a diminution or loss of one or both knee-jerks; or he may exhibit considerable static ataxy and difficulty in walking with his eyes shut, but not when open, or in co-ordinating his muscles so as to describe a circle with his toe. With these symptoms there may be more or less pupillary paralysis, and yet the patient may be unaware of his condition until the development of severe pains, gastric or laryngeal crises or loss of vision calls his attention, and that of his physician, to the fact that he has locomotor ataxia.

In fact, in these cases where sensory symptoms are late in making their appearance, the sclerosis of the posterior columns has usually made considerable progress before the patient becomes aware of the motor and other disabilities. Such symptoms as I have just mentioned are not likely to attract the attention of the patient, and, therefore, it can well be that a patient like the one in the case I have just described may be ataxic for a long time, until an accident or some other existing cause has occasioned the outbreak of sensory symptoms. This last fact the outbreak or increase of symptoms in a pre-tabetic individual after an accident—is one of considerable importance, and has a decided bearing upon this question. There is strong evidence for believing that a trauma may increase or hasten the development of the symptoms in a latent case of tabes of the kind above referred to. I use the term latent in the sense of a pre-existing tabes, in which the symptoms are confined to loss of knee-jerks, Romberg's symptoms and similar physical signs not noticeable by the subject himself.

Cases have not unfrequently been observed where either pre-existing symptoms have become worse, or there has been a development of new symptoms following an accident.

The complication, also, of such a case, with a trau-

\* This case was left to arbitration by three physicians, who awarded \$8,000 damages.

matic psychosis, seems to favor the exaggeration of tabetic symptoms, along with those peculiar to the psychosis. Sometimes the symptoms become so mixed up, it is difficult to separate those due to the two groups, and correctly ascribe to the existing tabes its own peculiar symptoms. The same is true of other organic diseases of the cord. It is known that the symptoms of a spinal tumor or a syringomyelia may similarly develop after an injury, although such diseases must have existed long previously.

A good example of this influence of trauma has been recorded by Bernhardt. It was that of a railway engineer who, one month after a slight accident in which the etiological factor was principally a psychical shock, developed symptoms of both tabes and a traumatic neurosis. Careful investigation showed that the man undoubtedly was tabetic for a long time previous to the accident. There was no history of syphilis or other ostensible cause in the case.

The second case of my own illustrates the fallacy likely to arise when symptoms of tabes develop after an accident.

It was that of a man who, in March, 1889, fell backwards from the last step of a railroad car, in consequence of the train starting unexpectedly. He fell upon the small of his back. The physician who saw him immediately after the accident testified to having observed a black and blue spot over the sacrum. The patient was confined to his room for three or four days owing to the pain in his back which ensued. From this time he began to grow weak; he complained of a burning feeling in his back. He was unable to attend to any work. He went to England, but returned without improvement; his legs became weak. An examination by his attending physician about six or seven months after the accident showed that the knee-jerks were absent.

At the request of Dr. P. C. Knapp I examined him for the first time, about one year after the accident, and found symptoms typical of locomotor ataxia. This was complicated, in my opinion, by other symptoms, which I considered to be those of a traumatic psychosis. It is not necessary to speak further of these details here. Although several experts, including Dr. Knapp and myself, made what we believed to be a careful inquiry into his previous history, we were unable to elicit from him any account of symptoms (such as pains) indicative of a

tabes existing previous to the accident. The patient seemed to be honest and was apparently perfectly frank. He admitted having had gonorrhœa, but denied syphilis. I was asked by the counsel in the case to give an opinion that the posterior sclerosis in this case was caused by the accident. This I refused to do, but said that I was willing to testify that most medical authorities gave trauma as one of the causes of locomotor ataxia, and on this authority it might be assumed that the disease *might* have been caused in this way, but I would not testify that it was.

The case went to trial, and I was summoned on this understanding. While the trial was in progress I obtained another examination of the plaintiff in the court-room in the presence of the other experts for the plaintiff. On again questioning the plaintiff, he frankly admitted, without reserve, that he had suffered from shooting pains in his legs, of a typically tabetic character, for some six years previously, and he stated that he had not admitted them before because he had misunderstood the questions, and had supposed that these pains were due to rheumatism, and had nothing to do with the case. The other experts who were present were then satisfied, as I was, that the patient had been pre-ataxic before the injury. The counsel in the case was then sent for, and was told that we were satisfied that his client had had his present disease before the accident, and that the accident was not the cause of the disease of the cord.<sup>3</sup>

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<sup>3</sup> The subsequent history is instructive. A few days later, after I had retired from the case, the counsel called upon me and asked whether I would testify that the disease from which the patient suffered might have been aggravated by the accident. To this I agreed, provided that no claim was made by him that the accident was the primary cause. To this he assented, and I gave my opinion in court that this patient had suffered from locomotor ataxia before the accident, but that on the evidence submitted the disease was probably aggravated by the injury. I afterwards learned that after I had left the court-room the counsel took advantage of the fact and put in the claim, in spite of all the evidence, that the disease was caused by the injury. A large award was given by the jury. This case illustrates how difficult sometimes it is to obtain a correct history of all the conditions preceding the outbreak of a disease. A patient may mean to tell the truth, but he forgets much, misunderstands the meaning of questions, and often does not remember the existence of early symptoms until his attention has been directly called to them. Medical men too often accept the mere statement of a patient regarding his history which frequently extends over several years, and yet in court it is difficult, even with a whole host of witnesses, to prove what were the actual facts in the physical condition of a person at any given time irrespective of any question of opinion.

Reasoning of the above kind would have less weight if it had been once demonstrated incontestably that a trauma which has not caused a gross lesion of the cord, such as a myelitis or hæmorrhage, has induced a sclerosis of the posterior columns in a single case, and still more, if it had been proved to be a frequent cause of this disease.

If trauma is, in general, an unquestioned cause, then it might be said that symptoms of posterior sclerosis having developed after an accident without other apparent cause, the probabilities were in favor of the trauma being the etiological factor in that particular case. But such reasoning cannot be applied until trauma has been proved to be an adequate cause in other cases.

It is necessary, then, to critically examine the evidence upon which the traumatic theory rests; but first one word regarding the kind of evidence which is essential to logically establish an etiological relation to a disease like tabes. It cannot be questioned that the conditions cannot be made too rigid. While it is true that if the etiological relation in general has been once established, it may be applied in individual cases on the doctrine of probabilities, such reasoning cannot be admitted to prove causative relations in general; or, in other words, the proof of a general law requires more extensive evidence than that of the applicability of a general law to a special case.

Therefore, in view of the fact that tabes can exist a long time unknown to the subject of it, and in view of the fact that symptoms of such a previously existing tabes may be awakened when not present by a trauma, it must be insisted upon, in examining the evidence afforded by alleged individual cases, that—

First.—The subject must have been proved free from tabes, either immediately before or immediately after an accident.

Second.—The subject must be shown not to have been exposed to other known causes, *e. g.*, syphilis.

Third.—The trauma must have been of a nature to produce a physical or psychical impression of an appreciable degree, and not such a one as people are frequently exposed to without suffering afterwards from tabes, *e. g.*, the extraction of a tooth or a mild bruise.

Fourth.—The symptoms must have made their appearance within a reasonable time after the accident; let us say one year.

Fifth.—The diagnosis must have been established beyond a reasonable doubt.

From this standpoint I propose to critically examine the evidence. As such an examination must necessarily be a digest of the cases thus far reported, and would prove wearisome to the general reader, I have thought best to insert this digest as an appendix to this article, and here merely give a summary of a careful examination of the cases. The quality of the evidence can only be properly appreciated by a study of cases themselves in the appendix.

TABLE I.

CASES INADMISSIBLE ON ACCOUNT OF TRIVIALITY OF INJURY, PRE-EXISTENCE OF SYPHILIS, LONG INTERVAL BETWEEN INJURY AND ONSET OF SYMPTOMS, DOUBTFUL DIAGNOSIS, ETC.

	Reporter.	Date of report.	History of syphilis.	Interval.	Remarks.
1	Schulze (case 2)	1867	not stated	Shortly after injury.	While pushing boat from shore with a pole, fell against side of boat. Indefinite symptoms for two years before accident.
2	" (case 3)	1867	"	2 years.	Fracture of leg.
3	<i>Sauitatz Bericht (case 5) f. der Deutsch. Meer., 1870-71.</i>	1885	"	3 months.	Wound in head by spent ball. Said to have suffered from severe disease of cord before enlistment.
4	do. lxxxii.	1885	"	2 months.	Tabetic pains in legs before the injury.
5	Leclorché & Talamon (S. & P.)	1881	Yes.	9-10 months.	Excision of corn from toe, followed by ulceration.
6	Sadler (S. & P.)		None.	12 years.	Wound near knee, preventing work for 15 days.
7	Lecoq (Petit)	1861	not stated	2 years.	Fracture of thigh.
8	Vulpian "	1879	"	15 years.	Amputation of leg.
9	Charcot "	1879	"	some months.	Fall from horse. No details.
10	Ball "	1868	"	a little while.	Resection of first phalanx of great toe for a corn.
11	Popenard "	1864	"	some months.	Contusion of knee from falling down stairs.
12	Popenard "	1864	"	10 years (Klemperer).	Fall or strain of back.
13	Edwards "		"	7 years.	Operation on a tooth.
14	Edwards "		"	2 years.	Operation for cataract.
15	Hirt . . . . .	1893	Yes.		Slid down a glacier.
16	Klemperer . . . . .	1890	None.	13 years.	Fall from horse, causing injury to foot.
17	Klemperer . . . . .	1890	"	16 years (?).	Gunshot wound of leg.
18	Prince . . . . .	1894	"	3 years.	Gunshot wound of arm.
19	Gee . . . . .	1886	"	1 month.	Crushed under heavy iron plate.
20	Straus . . . . .	1886	"	4 years.	Fracture of tibia.
21	Straus . . . . .	1886	"	3 years.	Fracture of elbow.
22	Straus . . . . .	1886	Multiple soft chancres.	3 months (?).	Fracture of patella. Said to have had weakness of legs before injury.

TABLE II.

CASES WHICH CAN NOT BE EXCLUDED, BUT WHICH, FROM VARIOUS CIRCUMSTANCES, ARE QUESTIONABLE AS EVIDENCE.

Reporter.	Date of report.	History of syphilis.	Interval.	Remarks.
1 Schulze (case 1)	1867	not stated (gonor-rhea).	Since accident (?)	Fracture of thigh. Examined 3 years after accident.
2 Schulze (case 4)	1867	not stated	Less than a year (?)	Slight injury. Also exposure to wet, cold and fatigue.
3 Spillman and Parisot.	1888	Denied.	4-5 months.	Slight injury to foot.
4 Leclorché & Talamon (S. & P)	1881	"	2 months.	Slight injury; fell upon edge of carriage, striking over hepatic region.
5 Bernheim	1888	"	"	Fracture of neck of femur. Imperfectly reported.
6 Prince	1894	"	2-3 months.	Fall from step of street-car; slight shock and sprained wrist. Slight injury.

TABLE III.

CASES APPARENTLY CAUSED BY TRAUMATISM.

Reporter.	Date of report.	Syphilis.	Interval.	Remarks.
1 Clarke	1866	not stated	Immediately (?)	Clarke did not see the patient. No record of physical examination at any time. R.R. accident. Meningo-myelitis.
2 Clarke	1867	"	1 month.	Fell while entering railroad carriage in motion. Struck back heavily; unconscious; bruised. Organic injury (?)
3 <i>Schütz</i> (case 1) <i>Ber. der Deut. Heer.</i> , 1870-71	1885	"	2 months.	Kick in left hip, causing contusion.
4 do. (case 2)	1885	"	11 months.	Gunshot wound in left knee, injuring peroneal nerve.
5 do. (case 3)	1885	"	3 months.	Gunshot wound of left elbow. Suffered from cold, wet and exertion.
6 do. (case 4)	1885	"	3 months.	Gunshot wound of upper arm.
7 do. (case 6)	1885	"	1 day.	Bruise of tibia. Cold, wet, and over-exertion.)
8 do., case lxxviii	1885	Not known.	1 year (?)	Severe wound of head by piece of shell.
9 do., case lxxx.	1885	Denied.	10-11 months.	Gunshot fracture of leg.
10 Horn (Petit)	1844	not stated	Some months	Fall from horse.
11 Klemperer	1860	Denied.	Short time, less than 1 year.	Blow, not serious, on lower leg.
12 Klemperer	1890	"	6 months.	Bad fall from horse.

On analyzing the cases critically it will be found that they may be separated into three groups. In the first—

the largest group (Table I.), may be placed those cases which, from various circumstances connected with their history, such as the triviality of the traumatism, the known existence of syphilis, the long interval interposing between the injury and the outbreak of the spinal symptoms, etc., we may feel morally certain arose from some other cause than traumatism. These are twenty-two in number.

In a second group (Table II.) may be placed those cases which, while traumatism can not be excluded as the cause, still can not with safety be admitted as credible evidences of a traumatic etiology. These cases are six in number. They will be found in the appendix described with sufficient detail. I will merely add here, by way of summary, that in four of the six the injuries were of a slight character; in one the interval (from the report) was indefinite, possibly three years; and in the sixth, the result of the physical examination as given in the report is not sufficient to justify the diagnosis. In none did a physical examination, either immediately before or after the injury, show the subject was not tabetic.

In the third group (Table III.) may be classed those cases which, from the nature of the injuries and other circumstances, might not unreasonably be regarded as the result of traumatism. They are twelve only in number. A critical examination of them reveals several flaws in their histories. In not one, as in the cases of the other groups, did a physical examination at the time of the accident demonstrate that the patients were free from spinal disease at that time; in all but three no statement is made, or it was not known (one case) as to whether the subjects had been infected with syphilis or not.

In five of the group the injury was a gunshot or similar wound. In the cases in which no history of syphilis could be obtained (3), it is possible that the subjects were previously tabetic, and the outbreak only of the symptoms was hastened by the traumatism, or the outbreak at this time may have been only accidental. At most, then, of all the published alleged cases, we have only three of four to which no specific objection can be raised.<sup>4</sup>

In looking over the evidence afforded by these cases

<sup>4</sup>The hundreds of published cases in which no such etiology is present must not be forgotten.

as a whole, the following facts are noticeable: first, in not a single one of the cases collected was it proved that the subject was free from disease in the cord immediately before or immediately after the traumatism, and, therefore, it is not possible to absolutely exclude a pre-existing tabes in any single case. In many of them, the interval which elapsed between the date of the accident and the outbreak of the tabetic symptoms was so long, sometimes many years, that a causal connection cannot be traced with any reliability.

In a number (about one-third) of the cases, the injuries were of the most trifling description, so that it seems almost an imposition upon our credulity to ask us to assume that they could have had a sufficient effect upon the nervous system to produce a sclerosis of the posterior cords.

If we are justified in rejecting such trivial injuries as a cause, it would seem that these cases are instructive as illustrating the fallacy of assuming the dependency of a disease of the cord upon a previous traumatism, simply because the symptoms developed soon after the reception of the injury. In the cases where the traumatism was of a more serious nature, the connection between the spinal symptoms and the traumatism was no closer than that in the subjects of the trivial accidents; and it would seem by the same process of reasoning that we would be justified in assuming an etiological relation in one class of cases as in the other, and yet common sense would seem to teach that the extraction of a tooth, the wound of a finger, or a sprained ankle, are not sufficient cause for so profound a disease of the nervous system, notwithstanding the outbreak of symptoms soon after the reception of such injuries.

It is also instructive to dwell upon the outbreak of tabetic symptoms after an injury in subjects who at first were supposed at such time to be free from tabes and yet in whom a more thorough examination has revealed the existence of a previous disease of the cord. Such cases show the fallacy of too quickly assuming an etiological relation between a traumatism and after-developing symptoms of spinal disease.

In a majority of the cases (23) the reports are silent regarding the presence of syphilis. In some, this fact was confessedly not known, and in a minority only

In the German army reports a number of cases are rejected by the author for this reason.



was it at best denied. Also, it so happens that of the cases, twelve in number (Table III.), in which in other respects the evidence in favor of a traumatic etiology is the strongest, and to which least objection can be raised, in only three was syphilitic infection specifically denied. The fact that in some of the cases the traumatism was a gunshot wound is a matter of some significance when one remembers the enormous number of such injuries that occur in the course of a great war, and yet the small number of cases of locomotor ataxia that is known to occur after them.

Besides the evidence to be derived from analysis of the cases, there is another sort of evidence which is of considerable weight. In all tables given by different writers to show the etiology of this disease it is remarkable how small a number are ascribed to traumatism, and this notwithstanding the fact that the assumption of a connection is generally made upon the mere sequence in time.

Erb, in his collection of 281 cases, reports trauma as the sole cause in only one case. Nevertheless Erb thinks that trauma plays a rôle in the etiology in about five per cent. of the cases, as he has seen an outbreak of the first symptoms after an accident in several instances, but this rôle seems to be only that of hastening the outbreak of the symptoms. According to Erb's well-known views, not accepted by all authorities, syphilis is the true cause in about ninety per cent. of all cases.

In the single case which Erb thinks was due to trauma alone, he fails to give any details. Other writers give trauma as a rather more frequent cause, but yet, even taking the highest figures, it confessedly plays an insignificant part.

As to the importance to be attached to the statement of a patient regarding the presence or absence of syphilis, it may be mentioned that Fournier states that he failed to obtain a history of preceding syphilitic infection in forty-four per cent. of cases of gummatous ulceration of the throat. Without committing oneself to figures, I think all will agree upon the great frequency with which it is impossible to obtain a definite history of syphilitic infection in cases of other nervous diseases which are undoubtedly of a specific origin.

Therefore, it would seem that too great weight should

<sup>6</sup> Die Aetiologie Der Tabes.

not be laid upon the mere inability to obtain a specific history in cases which apparently are caused by traumatism.

I have passed over such objections as might be raised on the ground of mere coincidence of events or faulty diagnosis, notwithstanding that in such matters these possibilities are entitled to some weight.

Taking all the facts above collated into consideration, it would seem that the current view that locomotor ataxia may be caused by traumatism *per se*, irrespective of a direct lesion of the cord, is not sustained by the published evidence thus far adduced. If such a relation exists further evidence is required before it can be accepted.

It would seem to be more probable, aside from mere coincidence, that when a sclerosis of the posterior columns develops after a traumatism, the subject was already doomed to this condition, the process having already begun, and that the traumatism at most accelerated the development of the symptoms, and possibly of the anatomical process.

## APPENDIX.

### A CRITICAL DIGEST OF REPORTED CASES OF LOCOMOTOR ATAXIA<sup>7</sup> ALLEGED TO HAVE BEEN CAUSED BY TRAUMATISM.

Most of the more recent and standard text-books on nervous diseases assert trauma to be a cause of posterior sclerosis.

Gray accepts this ætiology, and states that he has seen one case that was unquestionably due to an injury, but he gives no details. Spitzka and Gowers likewise assert that the disease may be caused by traumatism. The former states that one case out of eighty-one could be distinctly traced to this cause, and the latter that he has known the symptoms to develop a few months after a fall from a horse, but each of these authorities likewise fails to give details that would allow us to deter-

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<sup>7</sup> It is possible that in the examination of the literature some cases may have been overlooked, but it is believed that the most important as well as the great majority of cases have been included here.

mine the value of his evidence. Dana, with laconic positiveness, simply notes trauma as a cause. Hirt speaks decidedly in favor of traumatism, and in evidence cites a case, that of a man *who was the subject of syphilis*, but who fell and slipped some distance on a glacier with great rapidity, but no bones were broken and no dislocation occurred. The first tabetic symptoms made their appearance "a few months later." It does not appear that in this case any examination for tabes was made before or immediately after the accident, and the pre-existence of syphilis is significant.

Turning now to more valuable evidence, that contained in monographs, we find considerable material at our disposal. Passing over for the moment the very earliest cases, Leyden, in 1863, observed a case which he attributed to cold, but since then it has been quoted by later writers as an instance of tabes following trauma. It was that of a man without history of syphilis. He had suffered from constant sweating of the feet. Before Christmas, in 1859, a bar of iron fell on his foot, injuring three toes. He plunged his foot into cold water and applied ice to the toes. There was a suppression of sweat in this foot, and one month later in the other also. From this time, that is, one month after the accident, lancinating pains developed, and, later, ataxia followed.

In 1866, J. Lockhart Clarke examined the cord of a man who died three and one-half years after a railroad accident, but he (Clarke) had never seen the case during life. The posterior columns were sclerosed, but the author thought that in some respects the anatomical changes differed from those of tabes. There was chronic inflammation of the meninges, and pathological changes in the brain were also found. The history which was furnished to him by the attending physician was very incomplete and brief. It was stated that, following the accident, there were no wounds or fractures nor material contusions, but the patient, having been previously an active, intelligent man, conducting with success a large business, began at once to suffer vaguely with pains down the back and in the head, though not of a very acute kind. He gradually, though very slowly, failed. Symptoms, apparently of locomotor ataxia, developed, but no account of any physical examination whatever was given. The general account of his condition following the accident is vague and suggestive that some coarse injury of cord or membranes was sustained—a meningo

myelitis. No statement is given regarding the existence of syphilis. In neither this nor in the preceding case of Leyden's was there any record of an examination (which was presumably not made) that showed that the subjects were free from disease of the cord previous to or immediately following the accident, and in Leyden's case the accident was trivial. Although these cases have been more than once quoted in support of the traumatic theory, sound reasoning requires that they should be excluded.

In 1867, Schulze, in a monograph on the etiology of tabes, reported four cases. One of these had had gonorrhœa, but no mention is made of the existence of syphilis in any of the cases. (Of course, in the earlier cases less prominence was given to syphilis as a cause than would be done to-day). It is worthy of note that one of Schulze's cases suffered from indefinite symptoms previous to the accident, and in none of them did an examination immediately before or after the accident show that the posterior columns were healthy.

The first case (fracture of the thigh) was seen for the first time three years after the accident. The first tabetic symptoms were darting pains, from which the patient was said to have suffered since the accident.

In the second case, there was pain in the loins shortly after the accident, later extending down into the legs and soles of the feet. In this case, the above mentioned indefinite symptoms were noted for two years previous to the accident. While pushing a boat from the shore with a pole, the patient had fallen, striking his abdomen on the side of the boat. The immediate symptoms were not severe.

The third case was a fracture of the thigh. The fracture healed in eight weeks, but the leg remained (as was natural) weaker than the other, and the patient felt to and fro pains occasionally. No other symptoms of tabes for two years.

The fourth case was that of a man who was thrown against the trunk of a tree, bruising his left hip and lumbar region. He was able to continue working without interruption. Several days after the accident the patient was exposed to wet and cold and the fatigue of marching. He then felt dull pains in the lumbar region, followed by pain and weakness of legs coming on gradually. The date of appearance of these pains in the legs is not given, but surely pain in the small of the back,

after a bruise in the same locality, cannot be considered as characteristic of tabes. At what time the tabetic pains developed in this case is uncertain, though it was presumably in the course of the following year. It might fairly be asked whether exposure to wet and cold and the fatigue of marching might not be as potent a cause as an accident so slight as not to force the subject to discontinue his work?

It would appear, then, that Schulze's four cases fall far short of meeting the requirements of sound criticism. Syphilis cannot be excluded in any of the cases. One of them was known to have had venereal disease, though not syphilis. None of them were known to be free from tabes at the time of the accident; one had suffered from indefinite symptoms for two years previous to the accident; the injury in two cases was trivial; the date of the first tabetic symptoms is uncertain in two cases (although in one of these probably two years elapsed before they appeared).

In 1876 J. Lockhart Clarke reported another case which has all the appearance on a superficial examination of having been the result of an injury. A man, while getting into a railway carriage which was in motion, was thrown heavily on his loins across the edge of the floor of the car. He was instantly dragged in by his companions. He was unconscious for a few minutes. He was much bruised, and laid up for a month. At the end of this period he complained of numbness in his arms and legs and some weakness. Later, tabetic symptoms followed. It is assumed in the report that the above mentioned sensory symptoms were those of tabes, but it is quite likely, considering the nature of the accident, that they were due to some other condition. No report of any examination is given that would show that no gross lesion was sustained, or that immediately after the accident the patient was non-tabetic; and no statement regarding the presence or absence of syphilis is made, so that on analysis this case fails to have the weight that it appeared to have upon a hasty consideration.

Petit, in 1879, published an elaborate monograph on the subject of the relation between traumatism and tabes. This monograph has been frequently quoted and seems to have had considerable influence upon other writers. The author collected from the literature a dozen cases of locomotor ataxia occurring in persons

who, while presumably healthy, had previously suffered from an injury of some sort. The injuries were of all kinds, from the extraction of a tooth and an operation for a cataract up to serious accidents, like a fall from a horse. The author does not hesitate to include two cases of Edwards, in one of which symptoms of locomotor ataxia developed seven years after an operation on a tooth, and in the other, two years after an operation for cataract. Among the cases are included the two of Clarke's which I have already cited. The rest of Petit's collection is made up as follows: There is a case of Lecoq's, in which the inco-ordination developed as late as two years after a fracture of the thigh, and another of Vulpian, in which the ataxia developed fifteen years after the amputation of a leg. These are surely too long intervals to make it safe to infer the relation of cause and effect.

There is also a case communicated orally to Petit by Charcot, namely, that of a young officer who fell on his back from a horse. Ataxia developed some months afterwards. Charcot thought that the disease was due to the fall, but no details regarding dates, previous condition of the patient, etc., are given. Another case cited by Petit was one which was reported by Horn as far back as 1844, when locomotor ataxia was scarcely recognized. A man fell from a horse, and was unconscious many hours; no symptoms ensued at the time, but "the winter following" he suffered from attacks of pains and cramps in the legs. Other symptoms of tabes followed.

No mention is made of syphilis by Petit,\* and no examination of the nervous system at the time of the accident is recorded to show the cord was healthy at the time.

This writer also does not hesitate to include a case (by Ball) in which lancinating pains occurred, a little while after the resection of the first phalanx of the great toe for a corn. Finally, Petit's collection is completed by two cases reported by Popenard as early as 1864.

The first of these was that of a man who fell down stairs, with the result of having a contusion of the knee(?). He felt some lumbar pains, to which he paid no attention. Some months later he was ataxic. Whether or not he had locomotor ataxia is uncertain from the meagre details given and the absence of such evidence as would be afforded by an expert examination at the present day. Syphilis is not mentioned.

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\* I have not been able to obtain the original report by Horn.

The clinical details of the second of Popenard's two cases, as given by Petit, are also so sparse that it is not possible either to form an opinion as to whether the subject really had a posterior sclerosis; or, if so, whether the disease was ascribed to a fall in 1849 or to a strain of the back from lifting in 1852. The latter event was twelve years before the case was reported (observed?). No mention is made of syphilis by Petit.

Notwithstanding this mongrel assortment of cases with which Petit's article is illustrated, the author exhibits commendable caution in the conclusions which he draws from them, and limits himself to affirming that traumatism has an unfavorable influence upon the disease," and hastens the outbreak.

It will be noticed that this more conservative view is very different from that which holds that a posterior sclerosis is originated by a traumatism. As Petit's article has been frequently quoted, it is important that the character of the evidence contained in it should be recognized.

Straus, in 1886, reported three cases which, in the author's view, were caused by traumatism. An examination of them fails to justify our accepting them as evidence, as has been done by Spillman and Parisot and Klemperer. In the first case the first possible tabetic symptom was slight pain at the site of an old fracture of the tibia, which had occurred four years previously. The fracture was caused by a stone falling on the leg.

In the second case, the first tabetic symptoms were hyperæsthesia of the fingers of the left hand, and cramps and pains in the legs. These developed three years after an accident. This was a fall upon the right arm, causing a contusion of the elbow.

In the third case, the subject had previously had multiple soft chancre, twenty-six years previously, and had complained of weakness of the limbs for some time before the accident. He, the subject, then slipped upon the pavement and fractured the left patella. Three months later, pains came on in the left leg. This case is surely too suspicious to be cited in evidence, and the length of time that elapsed in the first two should rule them out.

The St. Bartholomew's Hospital reports for 1886 contain the report of a case by Gee. The accident was a

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<sup>9</sup> The second part of Petit's article deals with the effect of traumatism on pre-existing tabes, a subject which is not strictly within the limits of this article.

severe one, so much so that it is probable that some gross lesion of the cord or membranes, such as a hemorrhage, occurred at the time. This supposition is emphasized by certain peculiarities in the tabetic symptoms, to which the report calls attention, as well as the circumstances of the injury. The sensory and ataxic symptoms began in the arms at the same time as in the legs, and the cranial nerves were not affected. The subject had been crushed under a heavy iron plate weighing 13 cwt. "He was doubled up under it, chin on to chest and belly on to legs. He was got out unconscious and with blood flowing from mouth and nose." No notes of any examination of nervous system while in the hospital is given to show whether or not a gross injury was sustained, nor any statement of the condition of the cord at the time of his discharge from the hospital, excepting that he was "well enough" to be discharged, which may mean anything. Neurologists will understand how easy it is to overlook grave spinal disease. One month after the accident tabetic pains came on. There was no history of syphilis.

Spillman and Parisot, in 1888, published an interesting paper in which they attempted to show an etiological relation between tabes and injuries that produce an irritation of the peripheral nerves. According to these authors it is not trauma *per se* which induces the secondary degenerations in the cord, but the latter are indirectly the result of the peripheral irritations of the nerves. After referring to the literature of the subject, the authors cite one original case and several others communicated to them or reported by other writers.

Their personal observation was that of a man in whom tabetic pains developed four or five months after a moderate injury to the foot, due to the patient's catching his foot in the crevice of a turn table. The injury was slight, the skin was uninjured, although the heel was bruised and there were some ecchymosis on each side. There was no history of syphilis.

The second case was one communicated by Sadler. In this case, also, there was no history of syphilis. The injury consisted of a wound in the region of the left knee which prevented the subject from working fifteen days. Twelve years later tabetic pains developed.

The authors also quote two cases reported by Leclorché and Talamon. A pedicure in cutting a corn on the plantar surface of the great toe made a little wound,



which was supposed to have been poisoned, as it supplicated for two months. The wound was painful and caused pain through the whole foot. The patient put her foot in cold water to relieve the pain for some hours at a time. Shooting pains in the cicatrix persisted and pervaded the whole leg. Nine or ten months later severe pains developed in right leg, and three years later inco-ordination. One can hardly accept an irritation of this nature as a sufficient cause of tabes, considering the frequency with which similar ulcerations occur without such disastrous consequences, and it would seem more probable that the ulcer of the toe was due to the trophic disturbances of a pre-existent tabes, and was of a nature similar to that of the perforating ulcer. This view is strengthened by the fact that the patient was syphilitic, a fact with which the authors failed to be impressed.

The second case reported by Leclercq and Talamon was that of a man without a history of syphilis. In 1867 he fell upon the edge of a carriage, the edge coming in contact with his body in the hepatic region. He suffered from violent pain in consequence, without loss of consciousness. There was no fracture. He returned to his work in five or six days. Two months later he had cramps in his stomach with incessant vomiting. This is supposed to be the first tabetic symptom. In the following year lancinating pains developed and crises of vomiting. A year later still ataxia developed.

No specific objection can be made to this case excepting, as in the personal observation of Spillman and Parisot, the character of the injury was relatively trivial, and in neither case was there evidence that the subjects were free from locomotor ataxia at the time of the injuries. Spillman and Parisot also quote two cases from Remak, the details of which are too meagre to allow them to be utilized. The authors, while denying that trauma *per se* is a sufficient cause, admit that a peripheral irritation alone cannot originate a tabes, but that another factor is necessary, and this factor is nervous heredity. Neither is sufficient in itself, and each requires the other. This would seem to be a comfortable theory, and embraces at least two assumptions, each of which requires proof. Undeterred by the long interval of time that has elapsed in some of the reported cases, they make use of this fact to assert that the real origin (some peripheral irritation) can thus escape observation, and

seem to imply that such an irritation is a more common cause than is usually supposed.

A case communicated by Bernheim to Spillman and Parisot is as follows:

A patient on March 25, 1887, had a fracture of the neck of the femur. On December 1, 1887, that is, about nine months after the accident, lancinating pains developed in both legs. No history of syphilis. It is unfortunate that the details are meagre, and there is no record of an examination of the nervous system made either just before or after the accident.

In the report of the German army for 1870-71,<sup>10</sup> six cases of locomotor ataxia are mentioned which were thought by the author of the report to be directly due to traumatism. Besides these, there is a table of 100 cases, in twelve of which traumatism was more or less prominent in the history. The author himself does not attribute very great weight to the injury as an etiological factor in these last twelve cases, but thinks that other causes, such as exposure to cold and wet and over-exertion, were more prominent in most of them.

Klemperer, in his monograph, to be presently referred to, accepts two of these twelve cases, viz., cases 80 and 82, as due to traumatic causes. It seems to me that with equal justice a third case, viz., No. 78, should be accepted as of similar value. This connection between the traumatism and the spinal disease in the remaining nine is not sufficiently close to make them of value as evidence, therefore I think the author and Klemperer reject them with justice.

We have, then, nine cases all told, which might be thought to have a traumatic origin. This is the most valuable group of cases that has been thus far reported, and deserves careful consideration. Of the nine cases composing it, four were the victims of gunshot wounds of the limbs; one had had a gunshot wound of the side; one was wounded in the head by a spent ball; one was similarly wounded by a piece of shell; one had a contusion of the left hip by a kick, and one a bruise of the tibia.

In five of these cases (Nos. 2, 3, 4, 5, lxxxii.) the first tabetic symptoms came on from two to eleven months after the injury. In the one in which the interval was two months (lxxxii.), the subject, according to one account, had had "lightening-like" pains before the injury, and,

<sup>10</sup> Published in 1885.

therefore, must be assumed to have had tabes previous thereto. This case is interesting as illustrating the development of pains after an injury in a previously tabetic subject, and brings into the light a possible factor in all such cases. In one (5) it was stated by a former physician that the subject had suffered from a severe disease of the spinal cord before enlistment. In none of the five is it stated that the patient was free from syphilis, and in none of them had any physical examination been made at the time of the accident to show the condition of the cord. All had suffered from gunshot wounds, injuries which it is well known do not ordinarily produce any such effect upon the nervous system. It is particularly stated that two had been exposed to wet and cold and over-exertion on marches. The four other cases seem at first sight to be of more value as evidence of a traumatic origin. One was that of a man who had been kicked in the left hip and suffered from a contusion in consequence. About two weeks after this accident he suffered from pain in the back and in both legs of a boring character. Three months later unsteadiness of gait developed.

The second of these four had a bruise of the right tibia. On the day after the injury slight pain in both legs and the left shoulder was noted. Six weeks later weakness of both legs. This man had been exposed to severe wettings, cold and over-exertion, upon which some stress is laid in the report.

In the third case a note is made of pain and weakness in the left leg immediately after a gunshot fracture of the same leg, which would seem to be a not unnatural consequence and one which everyone knows is frequent after such injuries. The first real tabetic symptoms, gastric crises, came on ten or eleven months later (a rather long interval), but no other tabetic symptoms came on for nine years. In this case alone is it stated that there was no history of syphilis.

In the fourth case<sup>11</sup> the subject was wounded in the head by a piece of shell and knocked unconscious into a ditch. Cerebral symptoms developed immediately on recovery of consciousness, such as light hemiparesis, etc. After the healing of the wound, shooting pains developed in the legs, as well as a girdle pain and formication, etc. A year or more later, gastric crises and other tabetic symptoms came on.

Although the four cases of this second group seem

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<sup>11</sup> Case No. 8 of Table III.

very suggestive, it should be remembered that it was known of only one of them that previous syphilitic infection had not occurred, and in the absence of any knowledge of the nervous condition immediately preceding the accident, the outbreak of the sensory symptoms, assuming that the pains described were those of tabes, is equally compatible with the view of a pre-existing incipient tabes, accelerated and augmented by the trauma. In the last of the four (No. 78) the cerebral injury was so severe that it is not easy to distinguish clearly from the report the symptoms due to tabes from those due to the cerebral injury in the absence of a definite physical examination, which does not seem to have been made, and if we date the tabes from the gastric crises over a year elapsed before their appearance.

In the third case syphilis is denied, and the only specific evidence against the traumatic origin is the rather long interval of ten or eleven months and the nature of the wound (gunshot fracture). There was, however, no physical examination made just before or after the injury to prove the subject was free from spinal disease.

By way of summary, then, it may be said that a study of these important cases reveals the fact that in only one can syphilis be excluded. Two cannot be admitted as there is evidence of pre-existing tabes or spinal disease. In none was a physical examination made at the time of the accident. Another possible cause, cold, wet and over-exertion, had existed in two cases.

In 1860 Klemperer published a somewhat ambitious monograph on the subject of the traumatic origin of tabes. In this monograph the author collected in a table most of the cases found in the literature up to that date. All these have been mentioned above. Besides these cases, Klemperer recorded four new observations from Leyden's clinic.

Case one was that of a man who received a blow on his right lower leg. For some reason there was loss of consciousness, but it does not appear from the report that the injury was a serious one, and the wound healed in a few weeks. He returned to his work perfectly well, but "after a short time" it was noticed that the right leg was weaker than the left, pains were complained of in the cicatrix, which became worse and spread to the other leg. Nine months later the left leg was said to be weak, and one year and five months after the injury

tabes was diagnosed. There was no history of syphilis or other venereal disease excepting gonorrhea.

The second case was that of a man who had a bad fall from his horse, which fell with him, in 1871. There was loss of consciousness, and an examination showed a severe contusion of the right foot and ankle. There was no fracture, but rupture of the soft parts and necrosis of bone followed. The recovery was slow, but the first tabetic symptoms did not appear until 1884, thirteen years after the accident. Surely such a case should not be included as one having a traumatic origin.

The third case was one of gunshot wound in the left lower leg in August, 1870. The bullet was not extracted. Immediately after the wound there was painful cramp in the calf. Four or five months after the injury he was well. Paroxysmal pains developed then in the wounded leg, running through the site of the wound to the hip joint; the date at which these pains appeared is indefinite. The pains returned after long intervals, especially in 1886, in which year tabetic symptoms, such as gastric troubles, developed. Syphilis was denied. Considering that the bullet was not extracted, it would seem that there was sufficient local cause to account for these early symptoms in the leg, and inasmuch, therefore, as the date of origin of the tabetic symptoms is uncertain, tabes itself not being diagnosed until 1888, eighteen years after the injury, there is hardly evidence to connect the spinal disease with traumatism in this case.

The fourth case was that of a man who fell from his horse and broke his collar bone. He was well for some months after, until at the end of six months, when tabetic pains developed in his shoulder, spreading to the neck. These pains increased. Two or three months later he was exposed to cold, after which he became much worse. Two or three years later still other tabetic symptoms developed. Syphilis was denied.

It was only the first and last of these cases which with any reasonableness could be ascribed to the traumatism, and yet in neither of them did any examination at the time of the accident show the subjects were free from disease. No other specific objection can be raised against them.

But if such cases are to be accepted as of traumatic origin, the following case of my own might with equal propriety be looked upon as of this nature.

The subject was a claimant for a pension from the Government for results of injuries received in the war. His history was as follows :

In 1863 he was wounded in the left arm. Before he was wounded he had had chills and fever for two months. Malarial attacks had recurred at intervals up to the time of my examination, April 7, 1886. Then he suffered from boring, piercing pain in the left breast and side. The pain ran up into the left shoulder back of the head and across into the right shoulder. He also suffered from girdle pain around his chest, and attacks of severe piercing, shooting pains in the right thigh and calf. In 1866, that is, three years after he was wounded, the pain first appeared in his left arm. At the time of the examination the usual symptom-complex of locomotor ataxia was present, the hand being ataxic as well as the legs.

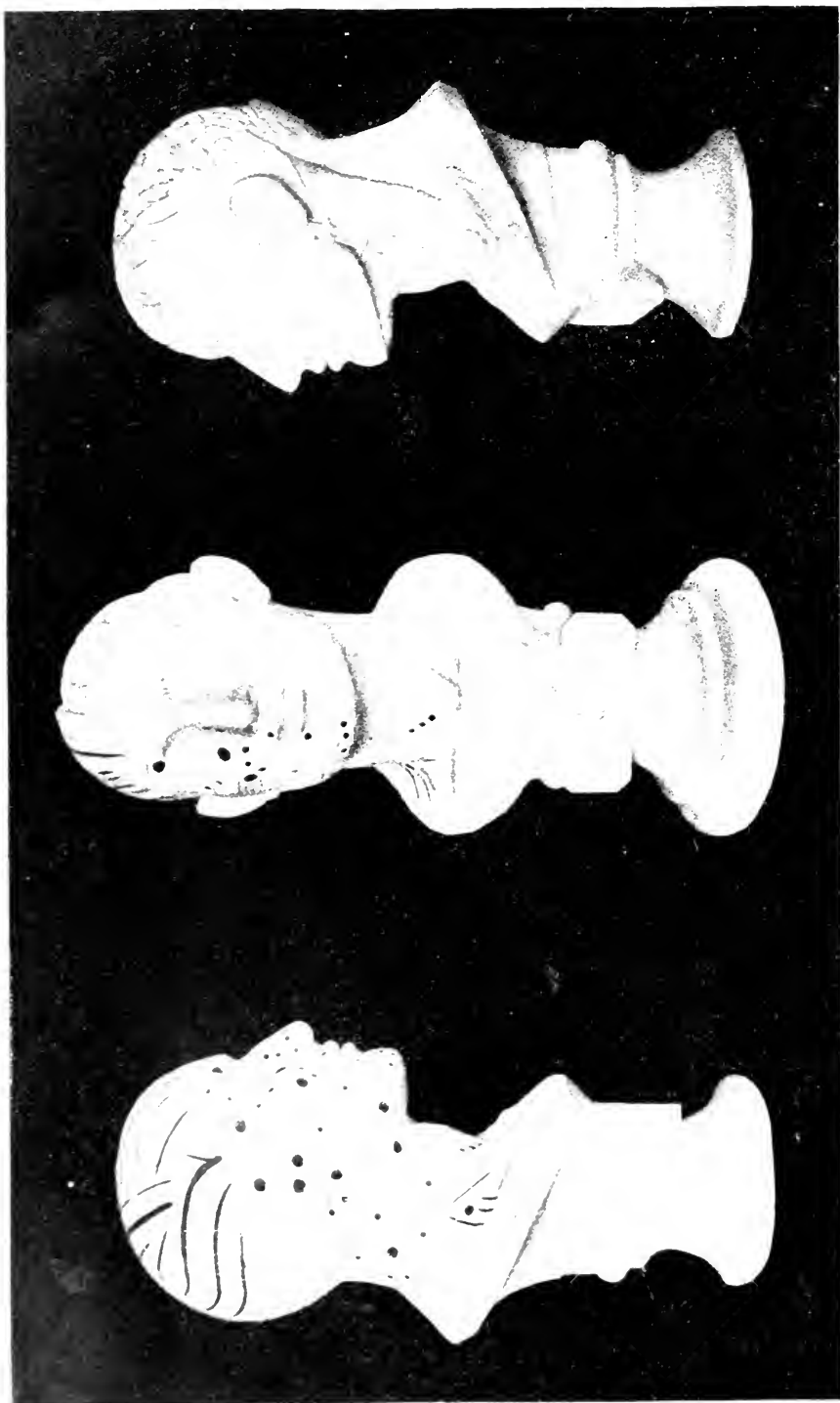
In this case I was unwilling to give the opinion that the locomotor ataxia was the result of a gunshot wound. The claimant denied ever having been infected with syphilis. Whether the existing malaria was an etiological factor in the case or not, is a question, it seems to me, worthy of investigation.<sup>12</sup>

**Stomach Diseases of Women.**—B. Robinson. (*Matthew's Medical Quarterly*, October, 1894.) Reflex neurosis in the viscera is nearly always from the genitals. The genitals are more liable to disease than any other viscera, and have the most intimate and profound nervous connection of all viscera. In reflex neurosis bowel troubles are chiefly secondary to genital disease. In gynecological clinical work nearly all cases who complain of stomachic disturbances were found to have well-established disease of the genital organs. It was also observed that as the genital disease recovered the stomach difficulties gradually decreased. The gradual steps in reflex neurosis are : (a) some point of irritation (genitals), (b) indigestion, (c) malnutrition, (d) anemia and (e) neurosis.

FREEMAN.

<sup>12</sup> See article by writer on the connection between malaria and degenerative diseases of the spinal cord in this Journal for October, 1889.





SEE PAGE 101.



## A NEUROLOGICAL BUST.

BY WILLIAM C. KRAUSS, M. D.,

Buffalo, N. Y.

AT the Seventeenth Annual Meeting of the American Neurological Association, held at Washington, D. C., September 22-24, 1891, I exhibited a bust,<sup>1</sup> which was designed particularly for the neurologist, as a means of ready reference in his consultation room, and for the teacher in neurology, as an important aid in the elucidation of facts in class-room demonstration. The features to which attention was called were the fissures of the brain represented by grooved lines on the head, and the electro-motor points of the muscles and nerves of the face, represented by grooved circles. Both sides of the bust were exactly alike.

I have recently improved the bust in several ways, and believe its value and efficiency to be greatly enhanced thereby.

The right side shows the relative position of the fissures and convolutions of the brain to the sutures and bones of the skull, the fissures being represented by grooved lines, deep or shallow, according to the size of the respective fissures in the brain. To render them more distinct and visible they have been traced in black. The principal sutures of the cranium are represented by ziz-zag lines traced in blue.

The face and neck present the electro-motor points of the muscles and nerves, the muscles being colored red, the nerves yellow.

The left side of the bust reveals the underlying anatomical structure, and herein does the new bust differ materially from the old. The skull cap has been removed sufficiently to disclose the left hemisphere of the brain *in situ*, with its fissures and convolutions fairly accurately portrayed.

The dissection of the muscles of the face and neck.

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<sup>1</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, Dec., 1891.

the arteries, veins, and a few nerves has been skillfully executed, and is the work of an experienced anatomical sculptor, aided by the advice of prominent anatomists, insuring the greatest amount of accuracy compatible with work of this kind.

Aside from the scientific worth and importance, the artistic beauty of the bust, as revealed in the plate, will be generally recognized.

#### CRANIAL REGION.

*Fissures.*—Sylvian fissure, central fissure (Rolando), parietal fissure, occipital fissure, superior frontal convolution (superfrontal), middle frontal convolution (medifrontal), inferior frontal convolution (subfrontal), ascending frontal convolution (precentral), ascending parietal convolution (postcentral), superior parietal convolution (parietal), inferior parietal convolution (subparietal), angular convolution, superior temporal convolution (supertemporal), middle temporal convolution (meditemporal), inferior temporal convolution (subtemporal), superior occipital convolution, middle occipital convolution, and inferior occipital convolution.

*Sutures.*—Coronal suture, squamous suture, lambdoid suture, and sagittal suture.

#### FACIAL REGION.

*Nerves.*—Trifacial nerve, superior branch; trifacial nerve, middle branch; trifacial nerve, trunk; trifacial nerve, inferior branch; hypoglossal nerve, accessorius nerve, Erb's point (supraclavicular point), phrenic nerve, brachial plexus, axillary nerve, infraorbital nerve, supra-orbital nerve.

*Muscles.*—Frontalis, corrugator supercilii, orbicularis palpebrarum, nasal muscles, zygomatic muscles, orbicularis oris, masseter, levator menti, quadratus menti (depressor labii inferioris), platysma myoides, hyoid muscles, sterno-cleido-mastoid, omo-hyoid, splenicus, trapezius, levator anguli scapuli, triangularis menti (depressor anguli oris), stylo-hyoid, digastric, risorius, and levator labii sup.

## REPORT OF THREE CASES OF BERI-BERI.

By F. X. DERCUM, M.D.,

Clinical Professor of Nervous Diseases, Jefferson Medical College; Neurologist to Philadelphia Hospital.

INASMUCH as beri-beri is so infrequently met with in this country, the writer believes that the following report, though incomplete, of three cases under his observation will not be without interest:

On December 7 a case was admitted to the nervous wards of the Philadelphia Hospital under the care of my colleague, Dr. Mills. On the same day a second case, and on December 11 a third case were admitted into the nervous wards under the care of the writer. All three were colored men, and had been working in the phosphate beds of the Island of Pitou Navassa in the West Indies.

CASE I.—A. S., aged 39, colored. American by birth. Family history negative. Has had syphilis and drinks to some extent. Went to dig in the phosphate beds in the Island of Pitou Navassa in the West Indies in May, 1894. He arrived in the middle of June in good health. He was obliged to work at hard labor, digging among the rocks and exposed to the sun. His food consisted of old salt beef, most of which was spoilt, poor bread, and occasionally beans. There was no fresh water on the island, the water supply consisting of rain water collected in a tank and teeming with animal life. The quarters consisted of a large wooden structure with bunks four tiers high, into which were packed 140 men. In the latter part of July the men began to take sick. All of them were compelled to continue at work long after they showed signs of œdema. The patient himself was seized with general weakness, headache, dizziness, marked vomiting and diarrhoea; also, about this time, he noted swelling, which began, he states, in the back of

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<sup>1</sup> Read at the meeting of the Philadelphia Neurological Society, December 17, 1894.

the neck, shoulders and face, and which next made its appearance in the legs, the arms, and finally the trunk. The abdomen especially was swollen. Marked pain and tenderness also made its appearance over the surface, the condition increasing until the whole of the body was enormously swollen. He attempted to continue at his labor, but was finally obliged to take to bed, becoming so weak that he was unable to walk. Later his condition improved gradually. On November 1, with forty others suffering with the same disease, he was brought to this country and landed at South Amboy, N. J., November 15. He was detained in temporary quarters until able to walk, when he came to Philadelphia and was admitted to the Philadelphia Hospital on December 7.

At that time the following condition was noted: The gait revealed considerable weakness, but it was atypical. There was no foot drop or "stepping gait," as is so often seen in alcoholic multiple neuritis. Patient complained much of pain in the ankles on attempting to walk. There was general œdema, most marked in the legs. It was, however, distributed all over the body, and could be demonstrated with readiness over the forehead, sternum and arms as well as in the legs. The abdomen presented the following interesting peculiarity: The epigastrium was excessively prominent or swollen, the swelling passing freely into either hypochondrium, but not extending as far down as the umbilicus. It appeared to be due to an excessive distension of the stomach. The skin and subcutaneous connective tissue of the abdomen were also decidedly œdematous. Pressure upon the surface of the body, grasping the arms and legs, revealed more or less marked tenderness. This was especially noticed in the calves. Tenderness was, however, not elicited in the nerve trunks. No marked impairment of cutaneous sensibility or of the temperature sense was noted. There was no analgesia. Grip was diminished. No wrist drop, no foot drop. The knee-jerk appeared to be normal. Because of the œdema of the face the patient's countenance presented a peculiarly smooth appearance.

Examination of the lungs negative; no increase in hepatic or splenic dullness; apparently marked distension of the stomach. Examination of the heart revealed a soft mitral systolic murmur, not transmitted. A soft aortic murmur was also noted distinctly, not transmitted. Eye report by Dr. de Schweinitz, negative.

Urine negative. An examination of the blood by Dr. Gaylord revealed a moderate degree of leucocytosis.

The œdema, which was evidently subsiding at the time of admission, steadily diminished. The distension of the epigastrium also grew markedly less until December 13th, when for some unknown reason it recurred so as to be again quite marked. During its recurrence the patient complained of increased weakness and of a marked sense of oppression in the epigastrium.

The value of these cases is much impaired by the fact that they were all decidedly convalescent at the time of their admission to the hospital, and during the time that they have been under observation a continued amelioration has taken place.

CASE II.—A. F. A., aged 27, Negro; birthplace, Arabia; occupation, seaman. Family and personal history negative. Syphilis and alcoholism denied. In May of the present year patient was under treatment in the Philadelphia Hospital for lobar pneumonia. In June he sailed to the West Indies, where he was employed on the Island of Pitou Navassa digging phosphate. His food consisted of salted beef, bread and beans. He was compelled to use for drink rain water which was filthy and dirty. After four months of hard labor his health began to fail. He first noticed an eruption on his arms and legs; this eruption he describes as being elevated, hard and dry, followed by desquamation and accompanied by considerable itching. This eruption ran its course in a few weeks, but hard nodules persisted in the skin in various situations. One and a half months after the appearance of this eruption, about the first of August, the abdomen and legs began to swell. This swelling was accompanied by considerable pain. No swelling occurred over the thorax, arms and head. The onset of symptoms was accompanied by marked weakness in the legs, necessitating his going to bed. According to the patient's statements neither foot drop nor wrist drop were at any time present. A more detailed history of his symptoms could not be obtained because of the great difficulty of communicating with him, the man speaking but little English, and being anything but intelligent.

Upon admission to the hospital, December 7, 1894, the following condition was noted: No œdema of legs or abdomen. According to the statement of the man, it had been absent for some forty days. Examination of the nerve trunks failed to reveal tenderness except over both

sciatics. Pressing or grasping the legs, thighs and upper arms elicited marked symptoms of pain. The abdomen appeared exceedingly painful to pressure. The patient also complained of pain over the left side of the head. There was also a slight cough, with a moderate amount of expectoration. The appetite was good; the patient stated that he occasionally suffered from nausea, but that he did not vomit. The bowels were constipated. He was able to get out of bed and to walk. His gait was merely paretic, being neither ataxic nor spastic; neither was it at all like the "stepping gait" so often noted in multiple neuritis due to other causes. Grip weak on both sides; K. J. apparently a little diminished. No atrophy of muscles. Cutaneous reflexes somewhat exaggerated. Tactile and thermal sense normal; no analgesia, no eye symptoms. Eye report by Dr. Schweinitz, negative. Examination of the other organs revealed the following:

Lungs.—Marked depression of supra and infraclavicular spaces on both sides the chest; feeble inspiratory movements; no increase in vocal fremitus; percussion resonance normal; no rales heard over chest.

Heart.—Impulse not perceptible. Heart sounds regular; soft mitral systolic murmur, not transmitted.

Liver and splenic dullness normal.

Urine.—Specific gravity, 1022; no albumin, no sugar.

Temperature chart shows slight rise above normal.

CASE III.—W. H., 21, Negro, American by birth; occupation, boat builder. Previous history negative, with the exception of syphilis four years ago. No history of alcoholism. Left Baltimore August 3, 1894, in good health. Arrived at the Island of Pitou Navassa August 23. After three weeks of severe manual labor, digging phosphate on a diet identical with that mentioned in describing the previous cases, the patient noticed that his feet and legs began to swell. The swelling was accompanied by marked weakness, while general soreness made its appearance over the entire body. The pain was especially marked over the anterior portion of the chest, and on one occasion he suffered markedly from dyspepsia. On two occasions he spat blood frothy in character and had a cough, which still persists. In addition, he suffered from headache, vertigo, nausea, vomiting, belching, and marked constipation. His symptoms improved shortly before leaving the island, but on No-

vember 6, while four days out at sea, the œdema returned, the feet, legs and face being swollen; the abdomen was exempt. He lost the use of his hands—became weak, as he expressed it, in his joints—and had burning sensations all over the body. Whenever he attempted the erect position he suffered from vertigo and palpitation of the heart. On one occasion, he states that he passed blood by the bowels. During this attack he noticed also that his urine was of a reddish color and that he had to void it five or six times at night. Upon admission to the hospital, December 11, the following condition was noted: Patient complained of aching pains in both feet and ankles, equally marked on both sides. Sometimes these pains were “jumping” (paroxysmal) in character. The patient complained of no subjective numbness of the legs or arms, but spoke repeatedly of burning sensations in the feet, especially the toes. When asked to leave his bed the following peculiarities of the gait are noted: Patient walks very cautiously, complaining of soreness in the soles of the feet. No ataxia or spastic character noted in the gait, no tendency to foot drop, no “stepping gait,” as observed in alcoholic neuritis. K.J.+, very slight ankle clonus on both sides. No wrist drop; grip diminished on both sides.

Examination of the nerve trunks revealed tenderness over both brachial plexuses, over the median, ulnar and musculo-spiral nerves of both upper arms and of the ulnar at the elbow and of the median and radial at the forearm. In the left arm this tenderness was less marked than in the right. Comparatively little pain is excited by gripping the arm or the forearm. Tenderness was found to exist in both sciatics, internal and external popliteals, and in the anterior and posterior tibials of both legs. No tenderness was elicited in the trigeminal distribution.

Tactile and thermal sense was found to be everywhere normal. Analgesia was, however, noted over the right leg, below the tubercle of the tibia, excepting a limited area just in front of the external malleolus, over the dorsum of the foot and over the ball of the foot. In the left leg analgesia was also found, excepting over the two malleoli. There was also marked analgesia over both arms and forearms. In other portions of the body the pain sense appeared to be normal. The muscles responded well to faradism, but this response was accom-

panied with pain and more or less cramps in the muscles.

Examination of the various viscera revealed the following: Heart—Impulse faintly visible in fifth interspace within the nipple line. Heart sounds regular, but diminished in intensity; no murmurs.

Lungs.—Slight impairment of resonance at right apex; increased vocal fremitus and resonance; prolonged expiration; vesiculo-bronchial breathing; no rales.

Liver and splenic dullness normal.

No œdema noticed in any portion of the body at time of examination.

Urine was acid; showed a specific gravity at 1011; a trace of albumin; no sugar.

Eye report by Dr. de Schweinitz negative.

Temperature chart reveals a temperature which on two occasions was decidedly subnormal. Pulse and respiration revealed no peculiarity.

Various forms of beri-beri have been recognized. In some cases the œdema predominates; in others the cardiac symptoms are in excess; in others still there is more or less marked atrophy of the muscles, and in a fourth group we find the symptoms evenly distributed, or in which the symptoms develop to but a moderate degree.

In Case I. the dropsical symptoms were particularly marked, while in Cases II. and III. no special group of symptoms predominated. Time will not permit us to enter into a discussion of the pathology of these cases, but it has been abundantly established by the researches of Scheube, Bälz, Pekel-Haring and Winkel that the nervous phenomena are directly referable to a neuritis. This neuritis appears at first to be degenerative in character, the changes being first noted in the myelin and axis cylinders, and later in the neurilemma, endoneurium and other connective tissue elements. The pain is doubtless to be referred to this neuritis as are many of the other symptoms. For instance, in cases in which heart symptoms have been very marked autopsies have revealed neuritis of the cardiac branches of the pneumogastric. The great distension of the stomach, which is noted by various writers and which was present in Case I., is also, perhaps, to be referred to a neuritis of the gastric branches of this nerve. Various pathological changes have also been noted in the spinal cord. These, however, very probably occur subse-



quently, and much later than the changes in the nerve trunks. Among the changes noted are degeneration and disappearance of the multipolar cells in the anterior cornu and proliferation of cells into the central canal.

Though wrist drop and foot drop seem to have been absent in the cases under consideration, Bentley has published a number of photographs showing both of these conditions occurring in beri-beri; and although in our cases the gait failed to reveal the high stepping character so frequently observed in multiple neuritis, Bentley has published a number of photographs in which this stepping quality is beautifully shown. It is therefore probable that in our cases these characteristics of gait and of the position of hands and feet were wanting, either because they are cases of moderate severity or because they are decidedly convalescent.

The cause of beri-beri is still an open question. Quite a number of investigators have conducted bacteriological researches, but their results lack uniformity. This is true not only of the researches collectively but of some of them individually. Thus it is exceedingly probable that the cultures obtained by Pekel-Haring and Winkler were impure. Indeed, it is quite certain, as abscesses followed some of their attempts at inoculation, that they were dealing with cultures containing pyogenic germs instead of or in addition to the germs of beri-beri.

A general review of the subject inclines one to the view advanced by Glogner, Scheube and others, that beri-beri is really due to a micro-organism derived from the soil, an organism which is, perhaps, like the hematozoan which appears to be the cause of malaria. Many of the circumstances connected with the etiology point to a paludal origin. If so, it must be an organism entirely distinct from that which produces malaria inasmuch as beri-beri is essentially an afebrile disease and also lacks that other great characteristic of malaria, periodicity. Further, quinine, so effective in malaria, is valueless in beri-beri.

## SUCCESSFUL USE OF GOLD IN TWO CASES OF NERVOUS DISEASES.

By J. ALEXANDER WADE, M. D.,

Danbury, Conn.

THE two following cases, successfully treated with arsenauero, are of interest from the unusual array of symptoms presented, their long standing and obstinate refusal to yield to any sort of treatment, and I consider it my duty to present them to your many readers for their consideration, with the hope that their history may enlighten the labor and relieve from an embarrassing position some professional brother who, like myself, becomes nonplussed in the treatment of such cases.

It will be noted that the first case in particular is unique in so many of its phases, the disease so tardy in yielding to the treatment varied so often by myself as well as by the many other physicians under whose care she has come from time time during the last ten years, and the complete recovery which followed the use of the remedy used makes it, I am confident, a valuable addition to the therapeutics of such cases.

Mrs. A., American, married, 34 years of age, the mother of one child, came to me with a history of extreme nervousness ever since the period of puberty, *i. e.*, since she was 14 years old. The menstrual function was unaccompanied by any irregular symptoms, nor could I find that the condition present was due to any diseases or reflexes, either from the uterus or ovaries, while a very critical examination of these organs showed them to be perfectly normal.

She said that she had had innumerable doctors, had tried almost all remedies, both regular and irregular, in spite of which she had gradually but surely grown worse. I found that seven years ago she had had an attack of paralysis, hemiplegia, since which time there have been frequent attacks of loss of sensation and hearing. She first came under my care in December, 1893,

and from a neurotic standpoint it was as pitiable a case as ever came under my observation.

After a very careful and minute examination, occupying several visits, I made the diagnosis of neurasthenia, complicated with a severe form of hysteria. The treatment was varied at different times, and I ran the gauntlet of nearly all of the remedies laid down in the text books for this complication. Nerve sedatives were given, such as valerian, asafœtida, sumbul, bromides, chloral, sulfonal, and the like, conjoining them with tonics like iron, arsenic, strychnia, phosphorus, the hypophosphites, etc., in many forms and combinations, while I also attended carefully to the clothing, diet, exercise and amusements, but at best I was only enabled to give my patient temporary relief.

During the following three months she developed many of the various motor, sensory and psychical phenomena, such as convulsive seizures, with retention of urine, hiccough, vomiting, while a phantom tumor of the abdomen existed, associated with flatulent distension of the intestines.

The sensory phenomena were loss of sensation on one side, usually the right, though sometimes the left, loss of taste, smell or hearing. At other times there would be extreme hyperæsthesia, with pain in the stomach and exquisitely painful abdomen.

There was also hysterical *clavus neuro-mimesis*, intense pain over the heart, and *globus hystericus*.

The psychical phenomena were lack of will power, very excitable, easily moved to tears or laughter.

During the attacks of the greatest severity she would compose readily, music, which was beautiful in its character, though when in her normal condition she did not know one note from another, and could hardly raise a tune. In this state she would repeat long poems, sometimes originating them herself, at other times quoting from the leading authors. A careful examination, when in her normal condition, revealed the fact that she knew nothing of these performances at the time when they were enacted, she could not make the simplest rhyme, nor did she remember of ever having read any of the many authors whom she so freely quoted.

About nine months ago, in consulting with Dr. Wile, of this city, in reference to this case, he recommended me to use *arsenauro*.

I commenced using it at once, giving my patient five

drops three times a day, which dose has never been increased.

The course of this case was uneventful. The improvement was apparent within a week after commencing the remedy, and my patient gained in health, weight and happiness, and was discharged, cured, about two months ago.

I have frequently seen her since, and she informs me that there has been no return of any of the symptoms: in fact, to use her own language, "Doctor, look at me; I'm as strong as Sampson."

My second case was that of a merchant, Mr. A., aged about 45, father of a family, all in fairly good health. He had long been suffering from tetany, and when I was called in, in August, 1893, I found the patient in a bilateral tonic spasm in both arms and legs.

In this case the usual remedies were exhausted, including the bromides, chloral, belladonna, hydrochlorate, hyoscyamine, etc., with baths, friction, careful attention to diet and secretion. The attacks would occur with intervals of from two to six weeks.

Three months ago I placed my patient on arsenauero, and since that time he has been free from all attacks. He has grown in weight, and he tells me he feels decidedly better.

In this case I commenced with five drops three times a day, increasing it to eight drops.

This case I have still under my observation, but am certain that arsenauero has given me by far the best results of the many drugs that I have used.

**The Gravity of Hysteria.**—Dr. G. Elliot. (*N. Y. Med. Jour.*, September 29, 1894.) Hysterical manifestations indicate an abnormal condition of the nervous system, and the condition will be aggravated if the patient is not properly treated. Prolonged or frequently repeated attacks may inflict permanent and serious damage upon the nervous system, and as a consequence a tendency to functional disorders of the nervous system may be transmitted to the children and grandchildren of the patient. Serious symptoms and even death may be caused by hysteria.

FREEMAN.

## Asylum Notes.

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By R. M. PHELPS, M. D.,

Rochester, Minn.

*The New York Lunacy Commission's Report* deserves prominent notice among matters affecting hospitals for the insane, partly because it is probably the most complete report made, and partly because it represents quite decidedly a new policy in this country, and one which has met with severe criticisms. Those who have read these criticisms will doubtless look with interest through its pages and expect to find a justification of its existence in so extended and full a report.

Let us enumerate some of the most important elements found.

We find, first, that the statistical tables of the various State hospitals have been reduced to a similarity of nomenclature so that the different findings can be compared. This we at once recognize as rational and sensible, even while those well acquainted with asylum work know that such tables are all of more apparent than real accuracy.

We find, moreover, that the statements of expenditures are all reduced to the same formula, and this also commends itself to prudent State management. We find again that they claim to have applied the principle of competitive examinations after adequate previous experience as determining who shall be the medical officers. This we recognize as relieving the service from the suspicion of favoritism in appointments. To also secure the desired chance of promotion to those holding subordinate offices, instead of allowing the appointments to go to outside inexperienced men. This also seems an advance, subject only to the criticism that the examiners should be able to take into account other qualities of the applicant besides mere scholarship.

We find still farther, that the commission has re-

quired monthly specified estimates, to be presented to them before the purchase of any articles.

This has been a cause of very extreme criticism. The right or wrong of this step must depend so completely on allowances made and the fact of any abuses existing previous to such control, that it is impossible to make accurate judgment without intimate knowledge. But, critically considered, it would seem that if a free resource is not allowed a superintendent to provide extra quality or quantity of food for the acute and curable cases, and to vary this according as his judgment dictates, that he is quite strongly handicapped.

We find again, that patients of chronic or harmless character, formerly in the poor houses, are being steadily gathered into the various state hospitals. We recognize the value of this in its elevation of the care of the chronic insane.

We find, then, that in nearly all of these points the effect of the control of this body has been seemingly good. But there is one point of view which leaves a feeling of disappointment, which may be mentioned. In all this long book we do not find if patients are more accurately or more carefully treated. Are the acute cases better treated? Is the treatment more individualized and minute? Is the routine broken up, is the hospital spirit taking the place of the custodial care? Are infirmary and hospital wards being established, and is the nursing spirit aiming toward that of the general hospital?

All of this may seem perhaps to some extent foreign to the report, but even if so it ought not to be. The business and economical tone is good, but medical aims and business aims will often diverge, and ought not the medical aim to predominate? The commission has seemingly worked toward the end of reducing all of the hospitals to a quite uniform and methodical routine of business methods. But it is very doubtful if the medical methods will be best subserved by being reduced to a similar uniformity.

The whole aim of modern progress in alienistic work is to put under the merely custodial aim, which has reigned so powerfully, and to instil the hospital spirit. But in order to foster the hospital spirit reports as hospitals, and of the clinical progress and of prominent clinical cases, and clinical, pathological and psychological studies would be most useful. If it be claimed that

this clinical report is foreign to the province of the Lunacy Commission, it is yet to be said that this commission invades the clinical work in its food and medical supplies, its accommodations, the distribution of officers, the management and its clinical tabular reports. Inasmuch as it thus invades the hospital as well as the business part, it should exercise great care and pervade the report with the true hospital spirit in the lines of research and study of the disease—insanity. If it is to hold the guiding reins, its report should be more fully enthused with the words of appreciation of clinical work done.

An autocrat is much better or much worse than a representative control, just in proportion as he is personally a more intelligent and more benevolent individual, or, on the other hand, is unadvanced and of political and selfish intentions. The Lunacy Board seems to have attained a quite autocratic position. We hope for the best final result from the exhibition of the broad and benevolent spirit in its control.

**Training Schools.**—The training school spirit, we note, is spreading, though not so rapidly, perhaps, as we might hope. Some schools do not accept unreservedly the hospital spirit in their work—a spirit which would make the school co-equal, as far as possible, with the training schools of general hospitals. This full-hearted commitment is earnestly commended. It commits the officers' work and their treatment to a full hospital spirit of work and thought, and tends to lead everything along in this line. With full appreciation of the routine of such hospital work it can yet be done. A short course of lectures to attendants may seem a respectable compromise, but it is a stopping short of the best.

Training schools were reported in the following hospitals for insane by T. B. Burr, who made a canvass in 1893. (Dates of organization are also given.)

Independence Hospital for the Insane, Iowa, 1880.

McLean Hospital, Mass., 1882.

Buffalo State Hospital, N. Y., 1884.

Essex County Asylum, N. Y., 1886.

Kankakee, Ill., 1886.

Willard State Hospital, N. Y., 1887.

Kingston Asylum, Ontario, 1888.

Middletown Hospital, N. Y., 1888.

Danver's Hospital, Mass., 1889.

St. Peter's Hospital for the Insane, Minn., 1889.  
Westboro' Asylum, Mass., 1889.  
Rochester Hospital for the Insane, Minn., 1889.  
Utica State Hospital, N. Y., 1890.  
Rochester State Hospital, N. Y., 1890.  
Eastern Michigan Asylum, Mich., 1890.  
St. Lawrence State Hospital, N. Y., 1891.  
Michigan Asylum for the Insane, Mich., 1891.  
Cleveland Asylum, Ohio, 1891.  
Toronto Asylum, Ontario, 1891.

He also notes the following as giving lectures, but as not organized or issuing diplomas: Retreat for the Insane, Hartford, Conn.; Western Pennsylvania Hospital, Dixmont, Central Indiana Hospital, Indianapolis; Asylum for the Insane, Hamilton, Ontario, and State Hospital for the Insane, Warren, Pa.

This enumeration makes about 24 out of 120 public State hospitals in the United States and Canada as having schools of lesser or greater rank. Although this represents a twelve years' growth, yet it must be noted as the initial and supposedly slower growth.

*The resignation* of Dr. C. B. Burr, of the Pontiac Asylum, Michigan, to accept the position of Medical Director of the private asylum at Flint, Michigan, is noted in its recent report. Dr. Burr was an active and popular man, and leaves much to the regret of the hospital authorities. Dr. Christian, after long and faithful service as assistant, receives the well-merited reward of the superintendency thus vacated.

*From the recent report* of the Elgin Hospital for Insane, Illinois, Arthur Loewy, Superintendent, comes the idea of leaving the patients' room doors open at night. This is also mentioned as in practice at the Willard State Hospital for the purpose of a closer relation of night nurses to patients, of avoiding the use of chambers in closed rooms, and of securing a sense of freedom, especially to the intelligent patients. One main advantage in Elgin seems to have been the compelling of night watches on each ward so opened. We do not understand that it is considered practical except to those who will not abuse the privilege too much.

*Bloomington Asylum* outlines a series of cases of general paresis in an instructive way. The cases are



very briefly given and the details are somewhat meagre, but they suggest what a very valuable means of accumulating and comparing experiences such a report might be made.

**Clark Bell** (*Bulletin of the Psychological Section of the Medico-Legal Society*, December, 1894) presents the opinions of 27 alienists, chiefly medical superintendents of hospitals for the insane, upon the questions: 1. Separation of business and medical work. 2. Manner of selecting superintendents. 3. Enforcing advanced scientific and pathological studies by law. 4. Degree to which patients' work should be utilized.

The answers show that of the 27, 20 are in favor of unconditional control of both medical and business work in such hospitals, 4 of the 7 others also wanted power over patients' diet, nursing, service, etc. Moreover, of the 20, 6 denied the wishing of more than enough power to control the treatment and life of the patients. This leaves 14 as in favor of unconditional power, and nearly all the rest in favor of controlling only the living, comforts, diet, nurses, and all inside matters.

Of the method of appointment 21 were in favor of present methods, while 4 ventured to assert the need of a method for ascertaining the scientific and practical attainments of the men. Concerning "enforcement by law" of advanced studies, 15 were in favor; 11 thought a law not necessary. About "employment" there was very little difference of opinion, all thinking it fully recognized and fairly well applied.

The answers show that nearly all the superintendents admit the value of having advanced neurological, clinical and pathological work, though some of them seem to see difficulties in the way of applying the ideas. The discrediting of their institution work is evidently felt, and the criticisms to an extent acknowledged, though some claim that they have previously made the same criticisms of themselves.

## Periscope.

### PHYSIOLOGICAL.

**The Mutual Relation Between Anæsthesia and Sensory Anæsthesia.**—Bechterew. (*Neurolog. Centralblatt*, 1894.) The clinical experience that disturbances of vision in the sense of optical hypæsthesia was frequently combined with anæsthesia of the eye, and that in such cases the impairment of vision was not hemianopic but strictly unilateral, i. e., confined to the anæsthetic eye, induced B. to study experimentally the relationship which exists between the sensory (for instance, optic anæsthesia, blindness, amblyopia) and the common anæsthesias of the corresponding organ of sense (anæsthesia of the eye to touch, pain, temperature, etc.)

For this purpose he performed section of the right ascending root of the nerve in the *med. oblong.* of a rabbit. Simultaneous lesion of the optic paths, which might have occurred if the third nerve had been operated upon in its peripheric course was then avoided. The effect was anæsthesia of the right side of the face, including the right eye. At the same time amblyopia of the right eye was observed. To what degree the vision was impaired and how it was manifested he does not state.

To explain the result of the experiment, B. calls attention to the clinical experience that cutaneous anæsthesia is frequently accompanied with contraction of the blood vessels, and consequently with ischæmia of the anæsthetic district, and that *vice versa* in ischæmic districts sensation is diminished. He concludes that the anæsthesia causes contraction of the blood vessels, and thereby disturbances of nutrition, and *vice versa*. Applied to the optic functions this means that the "usual" anæsthesia (anæsthesia of the eye to touch, pain, etc.) would lead to contraction of the blood vessels, and therefore to disturbances of nutrition of the parts intimately related to the function of vision (retina, optic nerve, etc.) The anæsthesia would also, by way of reflex, impair the action of the ciliary muscle, by which the visual functions would be still more diminished. Common anæsthesia of the organ of hearing would impair the auditory functions in an equal manner. (The functions of the stapedius muscle would be impaired by anæsthesia of the drum.) As the olfactory and gustatory functions are combined for specific and common sensation, the common anæsthesia would for this reason still more impair the sense of smell or taste respectively. ONUF (ONUFROWICZ).

**Parts That Do Not Grow Old.** (*Medical Record*, January 19, 1895.) Dr. Balfour says that the brain, if wisely used, largely escapes senile failure. Persons who think have often wondered why brain workers, great statesmen and others should continue to work with almost unimpaired mental activity and energy up to a period when most of the organs and functions of the body are in a condition of advanced senile decay. The reason is that the normal brain remains vigorous to the last, and its nutrition is especially provided for. About middle life, or a little later, the general arteries of the body begin to lose their elasticity and to slowly but surely dilate. They become, therefore, much less efficient carriers of the nutrient blood to the capillary areas. But

this is not the case with the internal carotids, which supply the capillary areas of the brain. On the contrary, they continue to retain their elasticity, so that the blood-pressure remains normally higher than within the capillary area of any other organ in the body. The cerebral blood paths being thus kept open the brain tissue is better nourished than the others.

FREEMAN.

**Galvanization of the Brain.**—J. F. Herrick, M.D. (N. Y. Med. Jour., September 15, 1894.) Dr. H. A. Hare has stated that it is impossible to pass a current of electricity, such as is usually applied to the head, through the brain by means of electrodes applied to the cutaneous surface of the head, because the current follows the course offering the least resistance, and with the electrodes applied to opposite sides, that the course is through the skin and soft parts external to the cranium. Dr. Herrick denies this, and says that from experiments made by himself to determine the resistance offered by bone as compared with other tissues (fresh beef, fat and fibrous tissue), muscle is the best conductor, and by far the poorest is fat and fibrous tissue. As the scalp is composed mostly of fibrous tissue and fat, we should expect to find it very resistant. It is alleged that in the percutaneous application of electricity the epidermis offers 300 times the resistance that all the intervening tissues do, and that the more succulent tissues offer the least resistance; therefore the brain would be among the best conducting tissues of the body. Dr. Herrick says the thickness of both sides of the skull together is little more than half an inch, and this with, say, seven inches of less resisting brain tissue, cannot offer a greater total resistance than about ten inches of skin and fascia through which the current must pass in going around the cranium. From experiments which he made to determine whether the current passed through the brain or not, it would certainly appear that electric currents may and do pass through the brain when an electrode is applied to either side of the head.

FREEMAN.

## PATHOLOGICAL.

**Contributions to Muscular Pathology.**—Fr. Schultze (*Deutsche Zeitschrift. f. Nervenheilkunde.*, Vol. vi., Parts I. and II.) Myokymie (muscular waving, especially in the lower extremities).

A healthy farmer, without hereditary taint, has been always well, excepting slight occasional attacks of headaches and diarrhoea. Following a physical overexertion the patient had to give up his work on account of painful spasms, tremor and tired feeling in the muscles of the lower extremities. On examination the somewhat voluminous muscles are in constant waving. Passive movements are accompanied by painful clonic contractions of the muscles of the calves and adductors of the thighs. Similar phenomena occur, though far less frequent, in the other muscles of the body. Occasionally, fibrillary twitchings can be noticed. Constant hyperidrosis of the lower extremities, even when naked, at a temperature of 16° R. The electrical examination of the gastrocnemius shows a tetanic contraction at a very weak current; otherwise the electrical reactions quite normal. The patient recovered under rest and the use of the prolonged warm bath.

Looking for analogies in the literature, the author finds similar cases reported by Talma in his paper, "Myotonia Acquisita," and by Kny in an article published in vol. xix., *Arch. f. Psych. u. Nervenkrankheiten*: "Ueber ein dem Paramyoclonus (Friedreich) nahestehendes Krankheitsbild."

The cases of Talma show some analogy to the muscular cramps occurring in cholera morbus and Asiatica, because two of the cases had suffered from diarrhoea.

In his case the author believes to find a cause in dilatation of the veins of the lower limbs  
FRANKEL.

**Contributions to the Pathology of Progressive Pseudo-Hypertrophy and Muscular Dystrophy.** Schultze (loc. cit.).

A review of eleven cases of muscular dystrophy, especially of the pseudo-hypertrophic type, which came under observation in the clinic of the author. In five cases the disease showed plainly the family character, occurring once in three brothers (two twins) and another time in two brothers. The disease began in the twins at the same time and had a different course, showing plainly the relationship of the different clinical forms of dystrophy. While one of the cases was the classical picture of a pseudo-hypertrophy, there was in the other a general atrophy, including some muscles of the face.

Two cases represented the form of juvenile dystrophy.

Regarding the muscles involved, the masseters were affected twice, once there was macroglossia, once the first interossei of both hands were atrophied, once hypertrophy of both scaleni antici majores. Once, a very rare occurrence, an extensive hypertrophy of the hand muscles. The electrical reactions proved normal in all the cases, excepting a partial De R. of the right deltoid in one case. As other points of clinical importance the author noticed a weak fixation of the caput humeri in the glenoid cavity and constriction of the deltoid muscles below the acromion.

Mentally, no marked changes were observed. Abnormalities of the skull and skeleton are to be seen quite often.  
FRANKEL.

**Paralytic Dementia.**—Henry J. Berkely (*Journal of Insanity*, January, 1895) reports on the pathological investigation of a comparatively recent case of paralytic dementia; that is, one that died in a comparatively early stage of the disease. He found no inflammation, but indications of the vascular origin of the trouble. He construes the course of the disease as follows:

1. A period in which the nerve structures begin to receive an insufficient supply of nutrient material from the blood, and in which the more active and recently acquired mental functions begin to fail; to which is added a certain degree of irritability, both muscular and mental.

2. A period in which the loss of nutrient material has become so pronounced that the starving tissues begin to feed on themselves; disturbed cellular metabolism results, which is clinically shown in the increased motor excitement and motor grandiose ideas.

3. A period in which the nutrient supply is so diminished and the tissue changes have become so far advanced that there is actual disintegration of the nerve cell and beginning overgrowth of the support substance, the stage of terminal dementia, and pronounced muscular paresis.  
PHELPS.

**Restitution of Pathologically Changed Ganglionic Cells.**—Proceedings of the Section of Nervous and Mental Disease of the Fifth Congress of Russian Physicians in remembrance of Pirogoff. Lubinow (*Neurologischeski Wiestnik*, 1894, p. 113).

L. starved a dog for a period of ten days in such a manner that it lost from 20 to 30 per cent. of its bodily weight. Then a trepanation was performed, a small piece of brain tissue removed and examined. Then the animal was fed again so as to make it return to its normal condition. In the period of refeeding pieces of brain tissue were removed for examination four, six and one-half and eight weeks after the end of the starvation period, and each time a new trepanation opening was made.

The specimens were hardened in two per cent. potass. bichrom., and stained with carmine hematoxylin, Ehrlich's mixture and Merkel's mixture.

In the piece removed at the end of the starvation period the following changes were observed:

Distinct transformation of the cell-protoplasm, part of which has disappeared altogether. The nuclei are badly (pale) stained. Around the numerous cells are leucocytes, which in some instances have entered the cell, thus filling the vacancies left by the disappearance of the protoplasm. The nuclei of the leucocytes are of a color similar to that of the protoplasm of the nerve cell. Size and shape of the leucocytes vary greatly; sometimes two or three nuclei are seen in them.

After refeeding of four weeks' duration, when the animal had returned to its normal physical condition, the brain tissue presented the following changes:

Signs of further decay and atrophy of cells. Of some cells only the nuclei are left, in others a narrow border of protoplasm is seen around the nucleus. The nuclei are now intensely stained and coarsely granulated. Sometimes nothing remains of the cell; its original position is marked by a vacuum. The number of leucocytes is increased; they are situated sometimes in the remaining protoplasm of the nerve cell, crowding the nucleus of the latter towards the periphery. Occasionally the nucleus is kidney shaped, in which case the leucocytes is situated in the "hylus." Sometimes the granula of the nucleus (of the nerve cell) are so coarse as to entirely hide the nucleolus from sight. Generally the protoplasm undergoes further retrogressive changes. The nucleus, after having gone through the described alterations, either returns to the normal state or perishes.

After six and one-half weeks of refeeding the following conditions were present: Around the nucleus a finely granulated and deeply stained protoplasm begins to accumulate; sometimes one sees the latter bulge out in the shape of sprouts. There are fewer leucocytes around the nucleus; none are seen around the protoplasm of the cells.

After eight weeks' refeeding the cells approach more nearly to the normal condition with the exception of the development of the protoplasmic processes, which still remain rudimentary.

Once the author by accident succeeded in seeing proliferation of a cell.

L. recommends that in studying the anatomical condition in cases of nervous and mental diseases the principal endeavor should be to find out the cause of the changes observed instead of only describing these latter, as by finding out the cause a regeneration of decaying nerve elements—that is, a cure of the disease—may be hoped for.

ONUF (ONUFROWICZ.).

## CLINICAL.

**Akinesia Algæra.**—Erb (*Deutsche Zeitschr. f. Nervenheilk.*, Vol. V., p. 6.).

Erb, in this article, continues the history of a remarkable case of akinesia algæra published in the same journal two years ago. The patient was a man who for twenty-two years had been a sufferer, and who for fourteen years had to preserve a strictly horizontal position in order to avoid "symmetrical" pain. He could not read, he could not tolerate being read to, and three or four words at one time was all that could be spoken to him at once without bringing on the pain. Mentally, he had remained fresh and clear and in a continuous and flowing manner. Under the influence of assurance and therapeutic measures directed to the heart and vascular system and the general nutrition, there was a marked improvement in the increased tolerance to hear speech, especially if it was not continuous. He was terribly frightened when people spoke quickly. There was no improvement in the general condition, and

especially weakness of circulation was not improved. "Symmetrical pain" in arms and legs, especially during digestion. The eating of meat caused this in the highest degree. When the first bite of meat reached the stomach this "symmetrical pain" and weakness became so great in the arms that a servant must needs help him carry the next bite to the mouth. Vegetables and soft foods were borne better. At the same time the condition of the stomach and bowels remained quite normal. An unlucky attempt to listen to rapid and uninterrupted speech gave him a severe setback, and then the severest "symmetrical pain" in the arms and legs was associated with pain in the head and noises in the ears.

From this time on it was sought to convince the patient that his pains were largely due to expectant attention and to engender the conviction that his symptoms were subjective psychical. And, moreover, that disturbance of the digestive organs was a weighty factor in causing the nervous symptoms, and that the strengthening of these was most important. Therapeutic measures to this effect were directed and weight given to suggestive self-influence, self-control and smothering of the expectant attention. The patient began by hearing two or three words spoken to him, with one or two extra ones added each time, and each succeeding time spoken the least bit faster, until he was able to listen to continuous and rapid speech. A similar plan was pursued to effect a change from the horizontal position which he occupied, beginning by raising his head a trifle and day after day adding a little more, until it was brought into erect posture by infinitely minute degrees; and this continued until he was able to walk and travel. The eventual results were astonishing.

In commenting on the case, Erb says that after he had seen this remarkable case and watched its course, and after a further experience with the disease, he inclines more to the opinion that *akinesia algera*, in spite of the fact that its clinical picture is sharply defined in many directions, is not an independent disease but a form of the functional neuroses in which the important symptoms are the psychical ones. As was the case when *agaraphobia*, *cephalic impressions*, *spinal irritation*, etc., were considered as individual diseases, so will it be with *akinesia algera*. After a more extensive and intimate acquaintance with it we shall learn to give it its right place in that large group of neuroses which now comprises *hysteria*, *neurasthenia*, *hypochondria*, certain *psychoses*, *maladie des tics*, etc., and determine how it differs from these other forms of disease.

J. C.

***Akinesia Algera.***—Bechterew (*Deutsche Zeitschr. für Nervenheilkunde*, 5 Bd., 6 Heft). The patient was a young man in whom disease began ten years ago after a trauma. A wagon drove over his feet. He was much frightened, lost consciousness for a while and had to stay in bed for a week. Since then pain in feet and legs, got easily tired, noticed lessening of sensation over the whole body; then pain in the muscles, especially when walking. Constant increase of these symptoms. Since two years in a gloomy mood, disinclination to work, apathy and frequent vertigo. Formerly had double vision.

His present condition is the following: Muscular action causes great pain in active muscles. This pain is accompanied by great fatigue, acceleration of respiration and pulse and flushing of the face. These symptoms are noticed when the patient walks or stands or presses one's hand, indeed with any muscular action; the muscles of the face being least affected. Percussion of the muscles, and also of the bones of the vertebral column and extremities, causes great pain accompanied by the other symptoms mentioned. Knee-jerk normal on the right knee, lessened on the left. Cutaneous reflexes considerably diminished. Marked analgesia over the whole surface of the skin. Pushing a needle deep in until it enters the muscles is painful, however. Electro-cutaneous sensibility, sense of temperature and touch diminished. Muscular sense im-

paired in all muscles; when one of his arms is passively raised (his eyes being closed) he cannot find it with his other hand; thinks his mouth closed, after it was continuously opened, while he had his eyes shut. There is also optic, auditory, olfactory and gustatory hypæsthesia. Looking into the light is painful and causes increased lachrymal secretion. Faradic excitability of muscles and nerves normal, galvanic qualitatively normal, quantitatively somewhat increased.

Bechterew considers the hyperæsthesia of the muscles, bones and articulations to irritation and insults of all kinds to be the most characteristic symptom of the disease. The latter is not a psychosis. In atremia, which in some respects resembles akinesia algera, tenderness of the muscles, bones and articulations to mechanical insults is not present.

ONUF.

**Amusia: Musical Aphasia.**—Dr. T. G. Edren, Stockholm (*Deutsche Zeitschrift für Nervenheilkunde*, 6th Vol., I. and II. Parts).

The author reports a case of transient paraphasia and word-deafness and permanent deafness for musical notes (*Fontaubheit*). At the post-mortem examination lesions were found in the anterior two-thirds of the first temporal, and anterior half of the second temporal lobe of the left hemisphere. Fifty-one other cases, bearing on that subject, scattered mostly in French and German literature, furnish the basis for the conclusions of the author.

The material is arranged in three categories as follows:

1. Aphasia, without amusia, twenty-four cases; four post-mortem examinations.
2. Aphasia, with amusia, twenty-two cases; five post-mortem examinations.
3. Amusia without aphasia, six cases; two post-mortem examinations.

Altogether fifty-two cases, eleven post mortem examinations.

The author believes that the data contained in the mentioned clinical and anatomical material justify the following conclusions:

1. Any pathological process within the skull destroys the musical faculties of the patient in the same way as the speech faculties, thus leading to different clinical forms of amusia.
2. The different forms of amusia have some clinical independence in relation to each other, as well as to the different corresponding forms of aphasia.
3. The clinical forms of amusia show a great analogy with the clinical forms of aphasia. The former are often, not always, accompanied by the corresponding forms of the latter.
4. Amusia may appear in the clinical picture without aphasia, and *vice versa*.

5. Some of the forms of amusia have very probably an anatomical independence, and are always localized in the neighborhood of the areas of the corresponding forms of aphasia.

6. Musical deafness (*Fontaubheit*), *i. e.*, the perception of notes, accords and melodies, without comprehending their musical meaning, is probably located in the first and second left temporal lobe, anteriorly to the area of verbal deafness.

FRANKEL.

**Astasia-Abasia and Its Treatment.**—By F. Friedländer, M.D. (*Neurolog. Centralbl.*, 1894, p. 354).

Friedländer finds that in astasia-abasia there is not a loss of the conception of movements, but an inhibition of association in the transmission of the conception of movements into voluntary motion. This inhibition is produced by auto suggestion on the foundation of abnormally exaggerated suggestibility, which latter Friedländer finds to be the most marked characteristic of every hysterical disease. Friedländer thinks, accordingly, that the treatment ought to be suggestive, but is against

treatment by hypnosis, as this "artificial hysteria" would tend to still increase the abnormal suggestibility of the patient.

Friedlander recommends treatment by gymnastics. He begins with passive movements of the legs in the lying position, then lets the patient make active movements to which he opposes, gradually increasing resistance in order to enhance the motor power. These exercises are continued for weeks, until rather strong resistance can be overcome. Then the same exercises in a sitting position; here chiefly resistance to flexion of the hips and to extension and flexion of the knee. Subsequently the same exercises in upright position, the patient supporting himself by taking hold of the backs of two chairs, between which the patient is placed. The first trial is to stand without support. When patient has succeeded in this, preparatory exercises for walking are made: both feet are alternately put forward and backward, the thighs are flexed on the hip, then the leg extended on the knee and the foot flexed and extended. When the patient has learned to make these movements with sufficient strength and without support for several minutes, he can be persuaded to make the first trial at walking.

About twenty minutes ought to be spent daily for the exercises. On the average good results will be achieved within from two to three months.

ONUF (ONUFROWICZ.)

**The Etiology of Graves's Disease.**—By K. Grube, M.D. (*Neurolog. Centralbl.*, 1894, p. 179).

Grube inclines to the view that morbus Gravesii is to be considered as an infectious disease, or, better, as an intoxication subsequent to infection, the toxic agency working on the thyroid gland, or the central nervous system, chiefly the medulla, or on both together. The blood vessels are the bearers of the toxic agency. That the latter should just affect the vital centres of the medulla can be explained by the complicated distribution of the finest blood vessels in the vicinity of these centres.

The observation of a case of Graves' disease with rapid fatal course which he reports led the author to suspect the infectious nature of the disease. As further reasons in support of his theory, he puts forth the following points:

1. The endemic and hereditary history of goitre in connection with the fact that frequently the goitre has been in existence for years before the symptoms of Graves' disease set in.

2. The swelling of lymphatic glands accompanying the disease.

3. The proven heredity of Graves' disease.

ONUF (ONUFROWICZ.)

**A Case of Neuropathic Lesion of the Tonsil, Velum Palati and Uvula, Complicating Multiple Neuritis.**—B. F. Westbrook, M.D. (*N. Y. Med. Jour.*, November 17, 1894.) A man, aged 51, became exhausted from excessive toil and anxiety for the success of his inventions. The breakdown was accompanied by an attack of diffuse neuritis, the symptoms being severe neuralgic pains, numbness and paresis of left upper extremity; less pronounced but similar affections of the lower limb, edema and glossy skin, and intense neuralgia of the left side of the face; also an eruption in the left temporal and malar regions. This was followed by rapidly spreading ulceration of the left tonsil, which extended to the pillar of the fauces, velum palati and uvula. There was a mild febrile movement. The urine was hyperacid, and, moreover, it was acid throughout the twenty-four hours. Besides the symptoms relating especially to the lesion of the tonsil and palate, there were five others probably connected with the neuritis, but whose relations to the ulcerated region were such that they might fairly be considered as affording corroborative testimony as to the tropho-neurotic nature of the disease. These were (1) a difference in the size of the pupils, the left being swollen and not responding so readily to light; (2) re-



laxation of the small blood vessels, with a reddening of the skin (edema and transient flushing); (3) congestion of drum membranes and paroxysms of auricular pain; (4) throbbing of the carotids; (5) swelling of the gums and loosening of teeth. The locations of the lesions in this instance would suggest the implication of the spheno-palatine ganglion, either by inflammation of the perineural tissue of the ganglion itself or by way of reflexes participated in by the nerve fibres connecting it with the faucial, palatine, nasal and aural structures. Under treatment the progress was steadily toward recovery. FREEMAN.

**A Case of Multiple Neuritis** (alcoholic).—By Herman D. Marcus, M.D. (*Phila. Med. and Surg. Reporter*, June 9, 1894).

The case presented, in addition to the usual symptoms, pains, paralysis and anesthesia of the extremities, the remarkably low temperature of 92° for a period of five days. MEIROWITZ.

**Puerperal Polyneuritis.**—Lunz (*Deutsche Med. Wochenschr.*, November 22, 1894). The patient, twenty-four years old, developed, three weeks after confinement, difficulty in swallowing, dizziness and diplopia. This was followed by paræsthesia and numbness in the right hand, and later in the left arm and hand. Nasal twang to speech and fluids regurgitated through nose. Examination a month later revealed diplopia, slight deficiency in sixth nerve, paresis of left seventh, and slight involvement of right seventh. Marked paralysis of both arms and paresis of legs. Knee-jerks lost; sensibility slightly affected. Diminished electrical irritability to constant and interrupted currents, but not altered quantitatively. Recovery began two months after the onset of symptoms, and progressed slowly.

The author believes that the larger number of cases of polyneuritis occurring during the puerperium belong to the pyæmic and septic group, but that some cases occurring during or after pregnancy must be attributed to a cachexia. A third group of cases cannot be classed with either of the above, but the confinement must be postulated as a predisposing factor and simply facilitates the actual causes of the neuritis. Under these actual causes must be considered the shock of pregnancy and labor, the retention in the blood of incomplete metabolic products, depravity of the blood, etc. The writer believes puerperal polyneuritis to be more common than is generally considered. COLLINS.

**On the Etiology of Peripheral Facial Paralysis.**—By Dr. Rudolf Hatschek (*Jahrbucher f. Psych. u. Nervenk.*, XHL., Bd. Heft 1).

Of the 80 cases of facial paralysis treated during a period of three and one-half years at Nothagel's clinic, there were no less than 10 in which relapses had occurred. In addition to these 10 cases, 26 others, in which relapses had taken place, are cited from the literature. Of the 36 recurring cases, 17 involved only one side of the face; in 18 both sides were affected; while in one no definite statement is made with reference to the side affected. Of the 17 cases in the first category, relapses had occurred twice in 11 cases, 3 times in 5 cases, and 4 times in one case. Of the 18 cases of the second category, relapses had occurred twice in 12 cases, 3 times in 3 cases, 4 times in 2 cases, 5 times in one case.

The majority of these cases appeared to have been peripheral. In some, however, where there was a concomitant oculo-motor paralysis, a nuclear origin had to be assumed.

The fact of the frequent occurrence of relapsing facial paralysis on both sides proves to the author that, in these cases, there is a constitutional predisposition, rather than a local predisposition occasioned by a former attack. As regards heredity as an etiological factor in relapsing facial palsy, too much weight must not be placed upon it; still, its influence is not to be totally denied. In some of the cases diabetes, syphilis, the acute infectious diseases, as diphtheria, mumps, angina and influenza, appear to stand in a causative relation to the recurrent Bell's palsy. MEIROWITZ.

### *Laryngeal Paralysis in Chronic Nervous Disease.*

Permewan (*Brit. Med. Jour.*, Nov. 24, 1894).

The author examined laryngoscopically 34 cases of general paralysis, of which 3 were in the third stage, 9 were in the first stage, while 22 were in the varying periods of the second stage. He concluded: (1) That the larynx is not infrequently affected in general paralysis of the insane. (2) That this affects first and chiefly the abductors. (3) That this does not necessarily depend on the association of tabes with the more generalized disease, but is the direct result of the degenerative and inflammatory changes which affect the central nervous system in general paralysis.

In two cases of disseminated sclerosis the author found the laryngeal muscles affected, and in one case of bulbar paralysis he was able to watch the onset of adductor paralysis becoming absolutely complete, and the supervention on it of the affection of the adductors. J. C.

### *Two Cases of Syringomyelia with Crico-Arytenoidicus Posticus Paralysis and Atrophy of the Trapezius.*—Weintraud (*Deutsch. Zeitschr. f. Nervenheilk.*, Vol. V., Part 6).

Case No. 1.—Laborer, 26 years old. Healthy until age of 21. First symptoms at that time were vertigo and vomiting, and following this right side motor and sensory hemiplegia. After three months' treatment with mercury and iodide of potassium disturbance of motility passed away; the loss of sensibility continued. After this time complained of headache, and noticed the disturbance of sensibility of right arm and leg increased, and that he received injuries and wounds of the hand which caused no pain. The appearance of multiple fissures in the skin and severe oedema of the hand caused him to go to the hospital. Examination showed dissociated paralysis of sensation of the upper left quadrant and complete right side hemianesthesia, progressive atrophy of the left interossei muscles and beginning atrophy in the same muscles of the right hand. Atrophy of the trapezius on both sides. Paralysis of crico-arytenoidicus posticus on right side. Kyphoscoliosis. Increased reflexes. Pupillary inequality. The author considers the complete right-sided hemianesthesia hysterical. To explain the disease manifestations he places the lesion in the upper dorsal and lower cervical cord, and believes that the gliomatosis involves mostly the left anterior horn. The spastic phenomenon in the lower extremities bespeak encroachment on the lateral columns.

Case 2.—Laborer, 61 years old. Disease began three and one half years before, with pain in loins and hips, extending to thighs. Feeling of itching, of weakness in legs. Shortly thereafter inflammation of the right hand, great swelling, extending to the shoulder, for which incisions were made. This bettered, but was followed by a second attack. Weakness of hand after this. Since a year, some difficulty in speech, especially when he has not spoken for some time. Loss of hearing, left ear. Examination: Mutilation of right hand. Partial paralysis of sensation, right upper quadrant. Beginning atrophy of right trapezius. Right side paralysis of posterior crico-arytenoid muscle. Disturbance of speech. Evidences of spasticity; slight ataxia. The one sided mutilation, the partial paralysis of sensation confined to right arm, the non-involvement of the bone in the mutilation, the disturbances of sensibility, the appearance of spasticity, the fibrillary twitching of muscles speak in favor of syringomyelia, the seat of the lesion being the under portion of the cervical cord.

Both cases, the author thinks, point to an involvement of the accessorius nucleus, with a tendency to involvement of the medulla, as was indicated by the speech disturbance in the second patient. The appearance of atrophy of the trapezius simultaneously with the laryngeal paralysis points to disturbance of innervation in the outer and inner branches of the spinal accessory. The author considers that his cases

tend to confirm the results of Darkschewitsch's and Dees's anatomical investigations, who proved the unity of origin of the outer and inner branches of the spinal accessory nerve and its independence of the vagus-glossopharyngeal nucleus. J. C.

**Disseminated and Successive Gangrene of the Skin of Hysterical Origin.**—Bayet (*Ann de dermat. et de syph.*, Vol. V., No. 5, May, 1894).

The patient was a boy of nineteen, in whom disease began with a superficial sulphuric acid burn on the left forearm. The burn healed in twelve days. The patient was clearly hysterical. Two days after the accident the morbid phenomena began to appear. A diffuse redness was first noticed, of much greater extent than the spot, which afterward underwent necrosis; then a number of bright white points, isolated from each other, the first sign of epidermic alteration. These points later took a brownish tint, coincidently with the disappearance of the hyperæmia and a hemorrhagic focus found about them. The process may be arrested at that point, but ordinarily it goes on. The necrotic points united and formed a large superficial scab of an ochre color. The shedding of the crust left bare a superficial ulceration, which healed with difficulty. As a rule, a true cicatrix was not formed; the epidermis was regenerated, but thinner, less resistant, and deeply pigmented. The epidermic regeneration was often interrupted by the formation of sero-sanguinolent blebs. When the process invaded the dermis, and a true cicatrix was formed, it often took a keloid aspect. The initial lesion is then epidermic, of uncertain nature but clearly of trophic origin. The disease appeared on the left forearm, the site of the burn. The subjective symptoms were marked, but there was no loss of sensation except in the pharynx. Simulation was ruled out by a direct observation made by the author under the auspices of others. COLLINS.

**Optic Neuritis as a Sign of Brain Tumor.**—By William H. Wilder, M.D. (*Chicago Medical Recorder*, May, 1894).

The author discusses the relative value of optic neuritis in brain tumor in a study of 161 cases in which either an operation or an autopsy had been performed. He considers briefly the nature and pathology of optic neuritis, states the various theories of Græfe, Schmidt and Manz, Hughlings Jackson, Leber, Edmunds and Lawford, and indicates where these hypotheses are found wanting. He also discusses the value of optic neuritis as a diagnostic sign in cases of brain tumor, its worth as a means of localization, and its significance with reference to the nature of the tumor.

Of the 161 cases investigated, 90 comprise growths of the type of glioma and sarcoma, with their mixed forms. Optic neuritis was found in 74.3 per cent. of the cases which were examined with reference to this sign. Out of 104 cases with choked disc, 37 showed involvement of the cerebellum, whilst in 25 the motor convolutions were the seat of the neoplasm; 90 per cent. of the cerebellar tumors were accompanied by optic neuritis.

The author remarks the infrequency of one-sided choked disc. In some cases the neuritis was more pronounced on one side than on the other, and this, in the large majority of the cases, on the side corresponding to the new growth. W. is inclined to believe that the cause of neuritis of the optic nerve must be sought in the irritation of the nervous elements by the products of tissue change in the growth, causing a descending inflammation, or that they cause a direct irritation of the nerve through the medium of the fluids of the optic sheath. Wilder lays stress on periodical attacks of blindness as a clue to a possible intracranial growth. MEIROWITZ.

#### PSYCHIATRY.

**The Polineuritic Psychosis** (*psichosi polineuritica*). By R. Collello, M.D. (*Annali di Neurologia*, anno XII, fasc. vii.)

C. reports 33 cases of multiple neuritis, some personal, others taken from the literature. Eight cases are described very thoroughly. That which characterizes all the cases is the combination of mental disturbances with the symptoms of multiple neuritis. In some of the cases the neuritic symptoms predominated; in others the mental disturbances; while in a few both were equally strongly pronounced.

Motor disturbances are usually first noticed in the lower extremities. The gait becomes ataxic and high stepping; afterwards, frequently though not always, paresis or paralysis of muscles follows. Paralysis of muscles of the upper extremities are not so frequent as those of the lower extremities. Lesions of the facial nerves and of the nerves of the soft palate, larynx and of the other motor cranial nerves, as well as of the other sensory cerebral nerves, may occur. The motor paralysis is a flabby one. There is diminished or abolished response of the muscles to the faradic currents, and diminished response to galvanic currents or actual reaction of degeneration.

The sensory disturbances belong to the late symptoms of the disease. Pain in the atrophied muscles, tenderness to pressure along the course of the nerves, hyperæsthesias or hyperalgesias, hypæsthesias or hypalgesias, retardation of the sensory transmissions, are often met with. The thermic sensibility is, as a rule, unimpaired; sometimes, however, diminished or perverted. The superficial reflexes are generally diminished occasionally normal, or even exaggerated. Tendon reflexes nearly always diminished. Vaso-motor and trophic disturbances are frequent (glossy, squamous skin, transversely striped and brittle nails, swelling of joints, edema periarticular, bed sores, etc.) Frequently there are symptoms indicating disturbances of the whole organism; dyspeptic symptoms, obstinate vomiting, diminished secretion of urine, lessened sexual desire, menstrual disturbances, tachycardia, etc. The functions of the bladder and rectum remain nearly always normal.

Among the symptoms of the mental sphere the most frequent and most characteristic is the amnesia. This amnesia is general, intense and profound, and may be entirely confined to the most recent events, while events of greater distance are remembered and narrated with minute detail. This absolute impossibility to recall the present often singularly contrasts with the precision of answers, the logic of reasoning, and the correctness of conclusions. It is an amnesia determined by the loss of ability to call up reminiscences or remembrances; these latter are fixed and retained in the unconscious sphere, and revive at the time of the recovery. This amnesia conforms with the laws regarding retrogression of memory. The most recent impressions disappear first, the older ones later; the affective abilities disappear slower than the intellectual processes; the acquisitions which have become almost entirely organic (as old and every day habits) are preserved.

When recovery takes place, it conforms equally with the laws regarding the restoration of remembrances. Frequently the amnesia passes unobserved. Long, thorough and repeated examinations are often required to recognize it.

The amnesia is often accompanied by disturbances of consciousness, incoherence, and narrowing of the field of thought, or symptoms of exaggerated irritability of the psycho-motor sphere predominate. These are manifested by extreme excitement, frequently leading up to attacks of mania (*pazzia furiosa*). Very frequently modifications of character, anomalies in the sphere of the sentiments, deep "lesions" of mental personality, are observed. At the beginning of the disease, disturbed sleep, interrupted by frightful dreams, visions of animals (serpents, mice, cats) and apparitions (fear of brigands, apparitions of blood-like color) occur. The apparitions present themselves to the patient with great clearness and vividness.

The prognosis is influenced by the rapidity of the course of the disease (best in chronic cases), by the general condition of the patient, by

the possibility of removing the cause (alcoholism, arsenic, or lead poisoning, etc.), and by the gravity of the complications pneumonia, decubitus). The latter are often fatal.

C.'s article is highly interesting, and recommends itself for closer study, especially the portion devoted to the description of the mental symptoms.

ONUF (ONUFROWICZ).

**The Warding-Off Neuro-Psychoses** (*Die Abwehr-Neuro-Psychosen*). (An attempt at a psychological theory of acquired hysteria, many phobias and certain hallucinatory psychoses)—Freud (*Neurolog. Centralbl.*, 1894, Nos. 10 and 11).

Freud accepts Janet's theory that the prominent feature of hysteria is a division (spaltung) of consciousness, but believes that this division occurs secondarily, not primarily. He thinks, with Breuer, that the foundation and condition of hysteria consists in the existence of dream-like states of consciousness, the so-called hypnoid states. Conceptions arising during such states are excluded from associative connection with the sum of conceptions forming the normal states of consciousness. Accordingly, a division of consciousness arises secondarily to the conceptions originated during the hypnoid state forming a separate psychical group.

Freud contends that in certain forms of hysteria and similar diseases this secondary division is the consequence of a voluntary act of the patient. The latter has received a painful or disagreeable impression and endeavors to keep it off his mind, to exclude it from consciousness, to forget that he received it. By this act the "affect" which was connected with said impression is "transferred." This can occur in two ways:

1. It is transferred into the somatic sphere, an act which the author proposes to call "conversion." The discharge of the affect then occurs by way of hysteria fits.

2. If the ability for "conversion" is lacking the affect is transferred upon other psychical spheres, it attaches itself to other conceptions, giving them the character of compulsory ideas.

In most of the cases observed by Freud, the original painful or disagreeable impression arose in the sexual sphere and the compulsory idea started by it was in some way associated with the original impression of the sexual sphere, or was of such a nature that the previously developed "affect" could attach itself to it.

To illustrate his theory, Freud reports three cases, one of which may be mentioned. A young girl suffers from compulsory reproaches. When she reads of false coiners the idea takes hold of her that she has made false money herself; when she hears of a murder she begins to ask herself whether she has not committed it. Finally, the compulsory ideas get such a firm hold of her, that she accuses herself before her relatives of having committed these crimes. A sharp examination elicited the fact that by advice of a friend the patient had masturbated for years, and, being fully aware of the wrong she had committed, made constant self-reproaches without stopping, however, her vicious habit.

In a third series of cases the patient also tries to keep off the painful impressions received, and does it so successfully that he finally believes that he never received them. But the moment he succeeds in thus deceiving himself, he is in a psychosis which must be classified as hallucinatory confusion. This form of psychoses, which Freud proposes to call retention-hysteria, is illustrated by the following case: A young girl has fallen in love with a young man and believes in the return of his affection. Unwilling to be disappointed in her belief, she endeavors to construct facts which clearly prove his indifference towards her in such a manner that she need not give up her belief in his attachment. One day, after she had vainly expected his visit, she fell into a state of hallucinatory confusion, during which she lived in the happy belief that he is with her, that he returns her affection, that everything is as before, etc.

ONUF.

## Society Reports.

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### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting held at the Academy of Medicine, Tuesday Evening, December 4, 1894.*

Dr. EDWARD D. FISHER, President, in the chair.

#### TUMOR OF THE BRAIN.

Dr. CHARLES E. NAMMACK gave the history of a case of brain tumor, and exhibited a post-mortem specimen showing the location of the growth, which was a spindle-celled sarcoma in the left frontal lobe. The full history of this case was published in the *New York Medical Record*, May 12, 1894. He also reported the following case, and exhibited a specimen in connection therewith: The patient was a male, aged 23 years; Russian; a furrier by occupation. He was brought to the hospital in a state of profound coma and the only history obtainable was that for three days previously he had complained of headache and diarrhœa. At the time of his admission both pupils were symmetrically contracted and the right eye-ball protruded slightly. The urine, drawn by catheter, showed a trace of albumen. There was some paralysis on the right side. The man did not recover consciousness, and died six days after admission. At the post mortem the lateral ventricles were found to be very much distended with serous fluid, and a soft, smooth tumor was found, completely filling the third ventricle and the aqueduct of Sylvius, and blending with the optic thalamus on each side. Under the microscope the growth proved to be a glio-sarcoma.

#### TWO CASES OF CEREBRAL SYPHILOMA.

Presented by Dr. NAMMACK.—The first patient was a man, aged 34; a cloth examiner. He contracted a

chancre in October, 1889; six weeks later he suddenly became unconscious and has no recollection of what occurred during the succeeding forty days. Following this there was hemiplegia on the right side, which confined him to bed for three months. The patient was put on specific treatment as soon as the initial lesion was discovered, and this was vigorously continued until the fall of 1893. About six weeks after treatment was discontinued the patient developed severe occipital headaches and bi-temporal hemianopsia, with ataxia and exaggeration of the knee-jerks. Under specific treatment these symptoms have now almost entirely disappeared. Dr. Nammack said the lesion in this case was probably a gumma situated in the substance of the optic chiasm.

The second patient presented was a man, aged 44 years, who complained of dizziness, bi-lateral occipital headaches, tinnitus, absolute deafness of the left ear, and diplopia. He also had the characteristic cerebellar gait. The history of syphilis in this case was rather obscure, but under specific treatment the man's symptoms have almost entirely disappeared. The diagnosis in this case was that of a gumma in the cerebellar region.

### A CASE OF MUSCULAR DYSTROPHY.

Presented by Dr. B. SACHS.—The patient was a young woman with all the characteristic symptoms of muscular dystrophy of the facio-scapulo-humeral type. On attempting to show the teeth, she is able to separate the lower lip slightly, the upper one not at all. There is also a loss of power in the eyelids, and the symptoms in the region of the shoulder-girdle are very characteristic. There is distinct hypertrophy of the infra and supraspinatus, and atrophy or partial atrophy of the serratus, the pectorals and the rhomboids. The patient is unable to raise her arm beyond a horizontal position. Dr. Sachs said that last July he excised quite a large piece of the infraspinatus muscle, and in doing so a considerable part of the nerve, as it enters the muscle, was accidentally removed. A histological examination of the specimen showed a great preponderance of fatty tissue; some of the muscular fibres were hypertrophied, but most of them were atrophied; there was also distinct evidence of a marked degeneration of the nerve. In view of the discussion that is now being carried on regarding mus-

cular dystrophies. the fact that the nerve in this specimen was found to be affected is of particular interest, although, of course, we cannot draw any great inferences from a single case. There has recently been a decided improvement in the condition of the patient. In the lower extremities there is slight atrophy of the muscles.

### NOTES ON RAPID GLIOSIS OF THE SPINAL CORD, WITH REPORT OF A CASE.

By Drs. L. E. HOLT and C. A. HERTER.—The case reported in the paper was that of a male child, aged one year, who was admitted to the Babies' Hospital on February 21, 1894. The family history was good. There were two other children, both healthy. Three months before the child was admitted to the hospital a weakness was noticed in the right arm and hand, and the head began to droop forward. The motor paralysis in the right arm gradually increased, and finally there was complete loss of power in both arms and in the shoulder muscles. The muscles in these regions became markedly atrophied and flabby in consistence. The muscles of the neck were exceedingly rigid, and the head was carried stiffly, slightly in advance of the normal position, with the face looking directly forward. There seemed to be some loss of power in the legs. The knee-jerks were exaggerated, especially on the right side. Ankle clonus was obtainable on both sides. There was considerable dyspnoea and coarse rales could be heard over the entire chest; there were no signs of consolidation. The child's temperature on admission was  $102^{\circ}$ ; it showed irregular daily variations, ranging from  $100^{\circ}$  to  $105^{\circ}$ , and even higher. There was occasional vomiting. The pupils were equal; they were normal in size and responded to light. There was partial analgesia of the body below the arms. The respirations ranged from 32 to 52 per minute. On March 16th slight strabismus was noticed, and the head was rolled continually from side to side. At times there was marked cyanosis. The temperature rose to  $106^{\circ}$ , and the child died at 6 P.M.

At the autopsy a grayish, projecting mass was found, which occupied almost the entire right side of the medulla, and a large part of the left side. The medulla was considerably enlarged from before backwards, and from side to side. There was also considerable enlarge-



ment of the cord, as far down as the eighth cervical segment, and from this point to the sixth dorsal there was some enlargement; below this the cord appeared to be normal. A histological examination of this growth, made at different sections of the cord, revealed a typical gliomatous structure, which involved, to a greater or lesser extent, the medulla and almost the entire cord. Dr. Herter said the appearance of the preparations examined renders it probable that the new formation of glia-cells were derived from the proliferation of the ependymal and peri-ependymal cells. Although the new growth involved almost the entire extent of the cord, it is only to the well-developed growth in the upper half of the cord that the symptoms presented by the patient are to be attributed. The high temperature of the patient could hardly be accounted for by the pulmonary condition present, and was probably due to the rapid destruction of nerve elements in the spinal cord.

In connection with the above paper, Dr. Herter exhibited a number of photographs showing the microscopical appearance of the growth at various levels of the cord.

#### REPORT OF A TUMOR OF THE LUMBAR REGION OF THE SPINAL CORD.

By Drs. IRA VAN GIESON and E. D. FISHER.—The patient was a man, aged 61; a native of Germany; by occupation a carpenter. He was admitted to the Hospital for Incurables on April 18, 1894, when the following notes were taken by Dr. A. F. Witmer, the house physician: Family history negative. Had syphilis at age twenty-five. No history of alcoholism. For about six months previous to his admission to the hospital, the patient suffered from pronounced dizziness on standing erect. His memory was good. There was diplopia of three years' duration. His hearing was good; speech normal; no gastric symptoms. During the past eight months the patient suffered from retention of urine, and frequent and severe attacks of diarrhœa. There was an entire loss of sexual desire. There was no weakness nor ataxia of the upper extremities, but he complained of progressive loss of power in the lower extremities, which commenced about one year ago. The patient never had any twitching or convulsions, nor hyperæsthesia; he com-

plained of a feeling of numbness from the waist down. His mental condition was unimpaired. Pupils responded both to light and accommodation; visual field normal. No paralysis of any of the cranial nerves. Co-ordination in the lower extremities is impaired; the patient is unable to stand erect and falls to the floor unless he has some support. There is no atrophy nor hypertrophy of the muscles. The gait is markedly ataxic, and the patient cannot walk at all without assistance. There was a gradual loss of sensation to touch and pain in both feet, more pronounced in the right. The knee-jerk on the right side was entirely abolished; on the left side it was normal. On May 22d the patient developed a large bed sore over the sacrum. From this time on he rapidly became weaker and died on June 3, 1894.

Dr. IRA VAN GIESON, who made the autopsy on the above case, reported the result of his histological examination as follows: The lesion proved to be an extra-medullary tumor of the spinal cord, cylindrical in appearance, and extending from a little above the second lumbar segment downwards into the cauda equina, a distance of nearly three inches. It measured three centimetres at its broadest part. It rested on the ventral surface of the cord, and at about the level of the fourth lumbar segment the pressure of it began to produce some distortion of the cord; at the fifth lumbar segment this distortion was very striking, both the anterior and posterior horns being pressed into a thin mass. In the sacral region the pressure effects were less marked. The extreme tip of the cord was found to be curled upwards on itself as far as the fifth sacral segment. Whether this retroflexion of the cord was produced by the growth, which, perhaps, grew from below upwards, Dr. Van Gieson said he did not know. The growth proved to be a glio-sarcoma. In connection with his paper, Dr. Van Gieson exhibited a number of charts and drawings of microscopical specimens.

Dr. FISHER stated that this patient was under observation for so short a time that a diagnosis of tumor of the cord was not made. The history of syphilis, the diplopia and the ataxic gait led the physician in charge to make a diagnosis of locomotor ataxia. Some of the characteristic symptoms of extra-medullary tumor, such as radiating pain from compression of the nerve roots, as well as pain in the back, were absent. It is to be regretted that the correct diagnosis did not suggest itself,

as it might have led to a more careful differentiation of the symptoms, and, perhaps, even to operative interference.

Dr. SACHS said it was unfortunate that the history in this case is so unsatisfactory, inasmuch as the growth was entirely extra-medullary. In tumors of the cord it is always an important point to determine whether the growths are intra or extra-medullary, because the latter are unquestionably much more appropriate cases for operative interference. He inquired of Dr. Van Gieson whether an operation would probably have been of any avail in this case?

Dr. VAN GIESON replied that he thought not. The growth was extremely vascular, and in certain parts of its course it was wrapped about the cord.

Dr. M. ALLEN STARR stated that one of the most valuable symptoms in the diagnosis of extra-medullary tumors of the cord is pain. In two such cases coming under his observation this symptom was very marked, and led to a correct localization of the growth. In both instances the pain was referred to a limited area covering the field of distribution of certain sensory nerves. In the first case, which was operated on by Dr. McCosh, a sarcomatous growth was found in the dorsal region. The patient went into a state of collapse and died ten days later of exhaustion. The second patient was a young woman who was operated on by Dr. McBurney, who found the cord compressed by an encapsulated tubercular deposit. The removal of this resulted within three days in complete relief from the paraplegia from which she had suffered; she was also able to perceive sensations in an area that had been previously anæsthetic, and regained some control over her bladder and rectum. This improvement continued for four weeks. She then had an attack of acute gastritis from eating some candy, and on the following day her temperature was very high and the symptoms of paraplegia again developed. They gradually increased during the next ten days; it was then decided to reopen the wound, and when this was done the tissues about the cord in that region were found to be so completely infiltrated with tubercular deposits that the operation had to be abandoned.

Dr. SACHS called attention to the fact that in the case reported by Dr. Fisher the growth covered the ventral surface of the cord; pain is only present when the growth is on the dorsal aspect or encroaches on the pos-

terior nerve roots. In the diagnosis of these cases we should not rely so much on the presence of pain as we should on root symptoms.

Dr. FISHER said that in the case reported the man complained of little or no pain. There were no shooting pains characteristic of involvement of any of the nerve roots. There was a gradual loss of power in the lower extremities, which became complete ten or twelve days before death, showing a compression myelitis.

Dr. HERTER said the diagnosis in these cases is often very difficult. Still, he did not see how the symptoms pointed to locomotor ataxia. The rapid progress of the disease, the lack of sensory symptoms, the motor symptoms, their unilateral character—these certainly did not point to tabes. The speaker agreed with Dr. Starr that pain is an early and very constant symptom in these cases of tumor of the cord; it is, perhaps, the most important symptom. Where the tumor involves the cord substance, we are apt to find a rather characteristic symptom, namely, marked contracture, either on the side affected or on both sides.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, December 17, 1894.*

Dr. WHARTON SINKLER, President, in the Chair.

Dr. F. X. DERCUM read a paper on

THREE CASES OF BERI-BERI. (See page 103.)

### DISCUSSION.

Dr. JUDSON DALAND.—This report has interested me, especially from an etiological standpoint. A review of the literature dealing with the cause of this disease has led me to the conclusion that the evidence is conflicting, and at present we cannot place our fingers on any one cause for beri-beri. It is quite evident that we are dealing with a polyneuritis. The disease known as beri-

beri exists in Japan, more especially in Tokio, in an epidemic form, but, as I have not had an opportunity to study the Japanese beri-beri, I restrict my remarks to those cases brought to Philadelphia. I am rather inclined to think that the cases under observation are examples of ptomaine poisoning. In the cases that I observed here a year ago we had evidences of overwork, semi-starvation, and they had been fed on decomposing fish. Dr. Dercum's cases had been fed chiefly on old salt beef which was putrid. The men received only a small quantity of vegetables, and the rain water used for drinking purposes was bad. It seems to me that decomposing fish, taken for three or four months under these conditions, might fairly be looked upon as a cause of the symptoms; and I feel that the cases reported to-night are precisely like the cases observed a year ago, namely, cases of multiple neuritis, the result of ptomaine poisoning from the use of decomposing flesh or beef.

The epidemics that occur in Japan may be due to other causes. The Japanese do not use much meat. They do use fish, but rice is the principal article of food. It may be that at times there is a fungoid change in the rice analogous to that which takes place in corn (*modnein nillagra*), and that the nervous symptoms are due to this cause. Ergotism from spurred rye is well known.

I was also interested in the cardiac lesions. Dr. Dercum described the physical signs as they were at the time. It is interesting to note that since the examination the mitral systolic murmur has disappeared. The murmur at the aortic orifice persists, and probably may be accounted for by an old rheumatic endocarditis.

The condition of the stomach is of interest, and even to-day this extreme dilatation persists. The supposition that it is due to involvement of the pneumogastric nerve is most plausible.

The cases of wet beri-beri, or those characterized by dropsies, seem almost always to be connected with a neuritis involving the cardiac branch of the pneumogastric, probably the trophic branches more especially, with consequent degeneration of the heart muscle, dilatation and cardiac failure.

The sensory symptoms referring to the skin are of interest, not only from the fact that in one of the three cases there was a considerable degree of analgesia, more particularly of the lower extremities, but from the fact

that this analgesia varied from day to day. Since the last examination there has been distinct improvement. The three cases I observed a year ago showed analgesia to a marked extent.

With regard to the blood, not many examinations have been made, but in all attention has been directed to the possibility of the presence of an organism either of the filaria or malarial group. In none of Dr. Dercum's cases did malaria exist, and the same is true of the cases seen last year. One fact that has been brought out in the study of these is that a leucocytosis of moderate grade is quite uniformly present. In all of Dr. Dercum's cases and in two of three cases I observed, there was an increase in the number of the white corpuscles. In one case there was a considerable number of microcytes, many so small as to present the appearance of mere dots.

It seems as though we are to be visited from time to time by beri-beri, and probably more frequently in the future than in the past. It is, therefore a disease requiring our attention, and should be carefully diagnosed from rheumatism, with which it is often confounded.

Dr. JAMES HENDRIE LLOYD.—I would ask whether or not there could be a scorbutic element in these cases? The diet is strongly suggestive of that. I saw these cases with Dr. Dercum, and was rather struck with the fact that the peripheral symptoms were not very marked. The knee-jerks were preserved, and the gait was not typical of peripheral neuritis. There is no doubt that in those cases there was the environment and the food which would suggest the possibility of a scorbutic element.

Dr. WHARTON SINKLER.—The question raised by Dr. Lloyd is an interesting one, for in nearly all the cases of beri-beri that have been reported there has been either an insufficiency of food or food which was lacking in nutritive qualities, especially albumin. This was so in the cases reported by Dr. Putnam of a number of sailors fishing on the banks of Newfoundland, where they had scanty food, consisting principally of fish, and lived under conditions which usually bring about scurvy. In Japan the epidemics seem to have occurred among those who lived almost exclusively on rice and fish. A number of cases have been reported as occurring on ships coming from the West Indies laden with sugar.

Dr. F. X. DERCUM.—So far as my reading goes scurvy as we know it can have no place in beri-beri.

Beri-beri is a disease which is well defined. It is a multiple neuritis due to a toxine or to some organism like that of malaria. That the disease is, however, not malaria is shown by the fact that it occurs where malaria does not occur, and that malaria is far more widely distributed than beri-beri. Further, quinine, as is well known, has no action in beri-beri. In Japan people in the best circumstances are often attacked by the disease, so that in many cases the ordinary etiological factors of scurvy are absent.

Dr. JUDSON DALAND.—In these three cases scurvy may be excluded. We know a good deal about scurvy, and I cannot recall a case of scurvy presenting beri-beri symptoms. In scurvy we do not have the multiple neuritis, although purpuric symptoms are common and the gums are early affected. Although from a dietetic point of view causes exist for the introduction of scurvy, yet the symptoms of beri-beri cannot be ascribed to scurvy.

## EXHIBITION OF PATIENT.

By Dr. J. MADISON TAYLOR.

Adjourned.

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**Epilepsy.**—By J. A. Larrabee, M.D. (*The Medical and Surgical Reporter*, Phila., Mar. 31, 1894).

This paper is mainly devoted to a consideration of the causes and treatment of epilepsy. L. considers heredity and syphilis as doubtful causative factors, but believes that chronic alcoholics and neurotics in general are apt to transmit epileptic diathesis to their offspring. He emphasizes the importance of a first attack of convulsions in children, and calls attention to the close relation of cause and effect between night terrors and epilepsy. He opposes the views of Henoeh and Goodheart that food is not a factor in the etiology of night terrors, and also thinks that the habit of taking food at night predisposes to night terrors. In his opinion, the bromide of ammonium is more efficacious than the other salts of bromine.

MEIROWITZ.

## Book Reviews.

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ANATOMIE DES CENTRES NERVEUX, par J. Dejerine, Professeur agrégé à la Faculté de Médecine de Paris, Médecin de Bicêtre, etc., avec la collaboration de Madame Dejerine-Klumpke, Docteur en Médecine, ancien interne des Hôpitaux de Paris, Lauréat de l'Institut de l'Académie de Médecine. Vol. I. General Methods of Study, Embryology, Histogenesis and Histology-Anatomy of the Fore-Brain. With 401 figures. Paris: Rueff et Cie. 1895. Price, 32 frs.

The anatomy of the brain has for many years been the field of predilection of wild semi-scientific speculation, and its reputation is that of a labyrinth of learned confusion, interrupted only by a few highways of well-ascertained fibre systems standing out of the otherwise marshy grounds. Descriptions by two different authors did hardly seem to refer to one and the same object, and Alexander Hill may have been somewhat justified in his cutting criticism of the anatomical literature on the brain in his "Plan of the Central Nervous System," in 1885. Still, it is perseverance and extension of the methods that he took to task which finally led to the first comprehensive, and none the less comprehensible, work on the human brain.

The work before us marks a decided departure beyond its worthiest predecessor, the latest edition of Quain's "Anatomy," inasmuch as it is dictated by the practical experience of a clinician and neuro-pathologist, besides giving the fullest account of the more strictly scientific, or, as many say, theoretical parts of the brain anatomy. This important feature characterizes every part of the book. The rich, perhaps unrivalled, pathological material of Bicêtre has given M. and Mme. Dejerine just that basis which anatomy of the nervous system needs in order to become a structure built on experience rather than on imagination and speculation.

The first part begins with the description and history of the methods used for the study of the nervous system (pp. 7-57). A very interesting discussion of the choice of a *post mortem* dissection, especially in pathological brains, with full description of the authors' own method, precedes the notes on hardening, embedding, staining and drawing. The second chapter (pp. 58-133) deals with the development of the nervous system, and contains valuable teratological remarks, besides a remarkably clear and well-illustrated account of the assiduous work of the last years.

The third and fourth chapters (pp. 134-232) are devoted to histogenesis, and to histology of the central and peripheral nerve elements in the adult.

The first part is profusely illustrated with drawings taken from



the publications of His, Retzius, Cajal and Ranvier; the selection of the drawings and their execution deserve equally high praise.

The second part of the volume treats the anatomy of the fore-brain.

The first chapter (pp. 233-386) covers the general morphology, the convolutions and fissures, the base of the fore-brain, and the configuration of its interior. Numerous drawings from photographs take the place of the customary diagrams.

The second and the third chapters (pp. 387-666) are practically a description of macroscopic and microscopic serial sections through the cerebrum, made in different directions, and forming the most complete atlas of those parts published so far.

A chapter on the cerebral cortex (pp. 667-741) and one on the white substance of the cerebral hemispheres (pp. 742-810) form the end of this first volume, rich in pathological observations with regard to the association systems.

The second volume will bring the description of the remaining parts and a systematic analysis of the fibre tracts.

The style is very clear, the current epitome on the margin of the pages very convenient. Schematic drawings are avoided; the figures are very accurately drawn, and clear.

Thus we have before us the first half of a work of fundamental importance for the progress of neurology, destined to bring the clinician into closer touch with the anatomical literature that is scattered in monographs and journals, and is here, for the first time, made satisfactorily accessible. Meritorious as the smaller works of Edinger, Obersteiner, Féré, Debierre, and the purely anatomical treatises are, none of them could give such a full account of the minute details that interest the clinicians and pathologists of to-day.

It is to be hoped that the second volume will soon follow and bring a very accurate index.

ADOLF MEYER.

URIC ACID AS A FACTOR IN THE CAUSATION OF DISEASE: A CONTRIBUTION TO THE PATHOLOGY OF HIGH ARTERIAL TENSION, HEADACHE, EPILEPSY, MENTAL DEPRESSION, GOUT, RHEUMATISM, DIABETES, BRIGHT'S DISEASE AND OTHER DISORDERS. By Alexander Haig, M.A., M.D., Oxon., F.R.C.P. Second edition, with 36 illustrations. London: J. and A. Churchill, 1894.

The appearance of a second edition of Dr. Haig's volume on uric acid in relation to disease is not only one of the many indications of a growing interest in the disorders of metabolism and general nutrition, but is also strong evidence that people prefer a bad book on an interesting subject to no book at all. For it must be said that the book before us represents a most unscientific attempt to fix the pathology of uric acid excretion and to refer a formidable array of diseases to the presence of an excess of uric acid in the blood or tissues. That this is a mild criticism of Dr. Haig's undertaking is shown in even a cursory review of its leading features.

In the first chapters of his book the author gives his views on the production and elimination of uric acid. Briefly stated these views are as follows:

First, the ratio of formation of uric acid to urea in the body is constant.

Second, since this ratio is constant, any deficiency in the amount of uric acid excreted by the urine must mean that there is a corresponding retention of uric acid in some part of the body.

Third, the urea produced in the body is never retained in excess, but is excreted promptly as it is formed; therefore, as the ratio of uric acid to urea in the urine is clearly a variable one, the quantity of uric acid in the body must constantly vary.

Fourth, any excess of uric acid in the body may be in the tissues or in the blood. If in the tissues, the uric acid may either (*a*) lie latent, as in the spleen, without causing symptoms, or (*b*) give rise to symptoms, such as the symptoms of gout, when the deposit is in the joints. If in the blood, the dissolved uric acid causes the most striking symptoms and is responsible for the numerous diseases whose description makes up by far the greater part of Dr. Haig's volume.

Fifth, the proportion of uric acid in the blood or tissues at a given time depends on the reaction of the blood; a reduction of the alkalinity of the blood causes a precipitation of uric acid in the tissues, an increase in alkalinity leads to the solution by the blood of the uric acid in the tissues, and favors the eventual elimination of the uric acid. Food, exercise, fatigue, perspiration, alkalies, acids, quinine, lead, mercury and a host of drugs owe their influence upon the organism to their effect in modifying the alkalinity of the blood.

Although these views have been more or less skillfully elaborated by the author, they form the basis, as stated above, of his conception of the pathology of uric acid excretion. Let us consider some of these fundamental propositions.

The statement that the ratio of uric acid and urea formation in the body is a constant one is without any foundation, and entirely ignores the extreme complexity of the chemistry of organic life, and the variations in circulating proteids, which render such an assumption in the highest degree improbable on *a priori* grounds. The burden of proof certainly rests in this instance on him who ventures on an assumption so removed from probability. The fact that in health the ratio of uric acid to urea excreted daily varies but little is of course no evidence whatever that the ratio of *formation* of these substances remains the same in all conditions of health and disease. The idea that the urea produced in the body is never retained in excess, but is promptly excreted, was disproved by the classical researches of Bright, Christison and Rayer in England early in this century, and the reviewer has definite proof from personal research that an excess of urea in the blood, amounting to 100 or 200 per cent., is no uncommon occurrence in nephritis, even when there are no symptoms of uremia. The recent careful work of Roberts affords no evidence that the precipitation of uric acid in the form of tophi of gout is due to an excess of acids in the blood, and furnishes strong reason for thinking that it is an excess of salines that is the leading factor in the precipitation of the urates. There is no foundation for Haig's statement that the administration of acids rids the blood of its uric acid by precipitating it into the tissues, and conversely, that the use of alkalies causes this precipitate to redissolve. To substantiate this statement, it would be necessary to make an elaborate series of experimental observations by means of the most perfected technical methods—an undertaking beset by great practical difficulties—and of such observations none are brought forward.

An excess of uric acid in the blood is the cause, according to Haig, of an increase of arterial tension, due to contraction of peripheral blood vessels. There is, however, no experimental evidence to show that uric acid dissolved in the blood has any such effect, while we have satisfactory evidence, especially that derived from the experimental work of Roy and Sherringham, that the chief chemical cause of contraction of the arterioles of the brain is increased alkalinity of the blood, while the in-

jection of acids into the circulation causes a relaxation of the vascular coats. Haig has by no means ruled out the influence of varying alkalinity of the blood in modifying arterial pressure. Even the proposition that the uric acid content of the blood increases with the blood alkalinity is far from being established.

The twelve chapters following those to which reference has been made are occupied with the application of Haig's physiological principles to the explanation of pathological processes. The variety of the processes that are attributed to the action of uric acid is shown by the titles of some of the chapters :

Headache; Epilepsy, Convulsions and Hysteria; Mental Depression and Fatigue; Asthma and Bronchitis; Dyspepsia and Gout of the Intestines; Raynaud's Disease; Paroxysmal Hemoglobinuria and Anemia; Albuminuria and Bright's Disease; Glycosuria and Diabetes Mellitus; Gout; Rheumatism and Morbus Cordis.

It would be useless, even if it were possible, to follow Haig's elaborate but incoherent argumentation for the causation by uric acid of the various diseases already referred to. It would also be quite useless to refute one after another his various "facts" and statements of opinion (some of which sound reasonable enough, but the majority of which are evidently mere speculation), for some of the most absurd views, born of the fancies of the moment, might require years of research to directly and scientifically controvert.

We have, however, another method, more radical and less difficult, of estimating Haig's claim for uric acid as a factor in disease. This consists simply in inquiring into the nature of the direct evidence that uric acid causes disease.

If uric acid in the blood, uricacidamia, is the cause of migraine, epilepsy, anemia, hemoglobinuria, albuminuria, chronic nephritis, rheumatism, etc., we should expect to find in the blood in these states an excess of uric acid.

The finding of such an excess would not, of course, necessarily mean that this excess stands in the relation of a cause to these diseases, for some other interpretation of the excess might be the correct one, but the point we wish to insist on here is that the demonstration of this excess in the blood is essential as a foundation on which to build an intelligent hypothesis of the uric acid causation of disease. The observations on the uric acid of the urine will not answer; they may be highly suggestive and doubtless are, but they do not give us positive evidence of the conditions of the blood as regards uric acid. Again, we have already seen that Haig's east-iron uric acid ratio theory is without any scientific foundation, and hence all the conclusions based on it are void.

What evidence, then, is offered us regarding the blood? The uric acid of the blood was estimated (pp. 46-52) in a case of cerebral hemorrhage, in one of cellulitis, in one of hemoptysis, in eleven cases with urate deposits in the joints, in eleven cases without urate deposits in the joints, in some cases of pneumonia, in a case of pneumonia with delirium tremens, in a case of morbus cordis, and in dogs and monkeys.

The figures obtained varied a good deal, and are too scattered to offer a basis (even supposing the figures to be correct) on which to generalize as to the influence of disease on the uric acid content of the blood. There are no observations on the blood in epilepsy, or headache, or nephritis, or on the greater number of diseases which the author refers to uric acid. Yet these results are garnished with suppositions in such a way as to make them prove almost everything the writer wishes. But this is not all. The technical means by which Haig reaches his results are open to the severest criticism.

In studying uric acid he employs the Haycraft method. This in the case of urine is admissible, for although the results given by it are high, the error is probably reasonably constant. In the case of the

blood, however, the error which may be introduced is enormous. For the Haycraft method is one in which the uric acid is estimated by the silver precipitate which not only includes what uric acid is present, but also other extractives which are present in the blood in relatively large and varying amounts. This method is, therefore, wholly unsuited to the use to which Haig, without any apparent appreciation of its character, has put it, and its results must be regarded as nothing short of worthless. But it is only right to mention the fact that Haycraft designed his method for the urine and not for the blood. It may be objected that it is a severe test to require the demonstration of the actual presence of an excess of uric acid in the blood as proof that such excess is related to disease in the manner claimed. It is a severe test, but nothing short of such an demonstration would constitute a basis for the views of Haig. Clever argumentation cannot take its place.

There is no evidence in the volume under consideration that the author has ever seriously considered the possibility that the presence of uric acid in the blood can be regarded as standing in any other relationship to disease than a causative one. It is the opinion of the reviewer, based on extended personal observation, that the excess of uric acid in the urine which is met with in disease is to be regarded as the result and expression of general nutritive disturbance or digestive disorder, and not as the cause of disease, except in a limited way.

It would be out of place to enter into this question here, but it should be noted that the clinical evidence, which points strongly to the correctness of this view, is reinforced by the studies of Horbaczewski as to the origin of uric acid and allied bodies. Horbaczewski's careful experimental observations indicate that uric acid is derived largely from the nuclein in the nuclei of the body-cells, especially the white blood-cells, and in consequence of their continual breaking down, and it is certain that uric acid, with its allied leucomaines, xanthine and hypoxanthine, guanine and adenine, can be obtained by the destructive transformation of nuclein. We cannot but consider it singular that this important work of Horbaczewski's, with all that it suggests, should have been not even mentioned in a volume dated 1894 dealing with the subject of uric acid.

The subject of uric acid in connection with disease is one where study is beset with difficulties, and it would be asking too much of any writer to make a wholly satisfactory book on it in the present state of our knowledge. We do not criticise Haig's book because it fails to settle the relation of uric acid to disease, but chiefly because of its pernicious method, the faulty observations it records, and the loose generalizations it puts forward. Such a book cannot fail to do harm by disseminating false yet plausible ideas, which few practitioners have time to investigate and to controvert for themselves. On the other hand, the book has probably done good by stimulating inquiry and discussion in a field of medicine which is most important from a practical as well as from a theoretical standpoint.

In conclusion, it may be pointed out that we must not take too narrow a view of the subject under consideration. An understanding of the rôle which uric acid plays in animal nature is intimately bound up with the more general problems of nutrition, with the subjects of digestion, absorption, the formation and destruction of the blood, and the metabolic processes generally. No progress can be made in our understanding of uric acid and its allied leucomaines without corresponding progress along these collateral and overlapping lines.

We believe the day will come, though it be distant, when some patient investigator, of the type of Darwin, with a vast array of facts before him, will study these facts in their relations to one another, and enable us to see order and obedience to the larger laws of pathology, where at present there is little but the confusion of seemingly unrelated facts.

C. A. HERTER.





Fig. 1

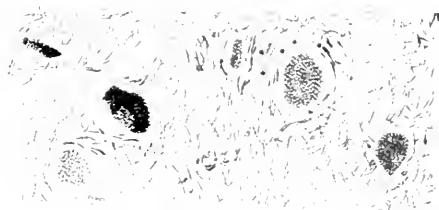


Fig. 2

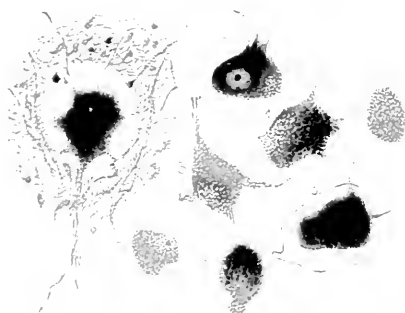


Fig. 4

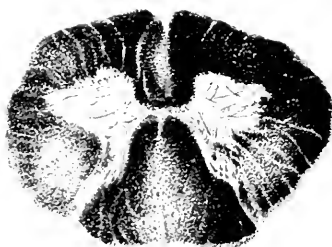


Fig. 3

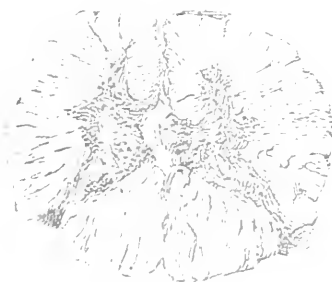


Fig. 5

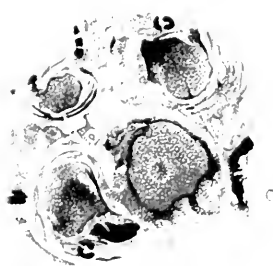


Fig. 6

THE  
**Journal**  
OF  
**Nervous and Mental Disease.**

**Original Articles.**

AMYOTROPHIC LATERAL SCLEROSIS WITH  
BULBAR PARALYSIS AND DEGENERATION  
IN GOLL'S COLUMNS: A CONTRIBUTION TO  
THE PATHOLOGY OF THE PRIMARY COM-  
BINED SYSTEM DISEASES.<sup>1</sup>

*(From Prof. Henschen's Clinic in Upsala, Sweden.)*

By LUDVIG HEKTOEN, M.D.,

Chicago.

MODERN histologists teach the existence within the nervous system of innumerable anatomic, functional and nutritive units or "neurons,"<sup>2</sup> each being a ganglion cell with its axis cylinder which, becoming a nerve fibre, end in free dendritic ramifications. Two sets of motor "neurons" are recognized, namely, the indirect of Kölliker (Waldeyer's of the second order), which consists of ganglion cells in the brain cortex, their axis cylinders which pass down in the pyramidal tracts and end in free branches around the cells in the motor cranial nerve nuclei and in the anterior horns of the spinal cord; the ganglion cells just

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<sup>1</sup> Presented to the Swedish Medical Society, Stockholm, November 27, 1894.

<sup>2</sup> Waldeyer and Kölliker prefer the word "neurodendren" for etymologic reasons. See latter's *Handb. der Gewebelehre*, Bd. ii., 1893, pp. 1 and 3.

mentioned, with their axis cylinders that form motor nerve fibres and end in the muscles, form the direct motor "neurons" of Kölliker (Waldeyer's of the first order).

The direct sensory "neurons" of Kölliker (Waldeyer's sensory neurons of the first order) are the large cells in the spinal ganglia whose T-shaped axis cylinders send one branch outward as a sensory nerve fibre and the other inward as an intra-medullary fibre. Full clearness has not yet been reached concerning all the higher, indirect sensory neurons, but it seems quite settled that Clarke's columns, the nucleus funiculi gracilis, and the thalamus are some of the centres for new systems (Kölliker, Waldeyer, Monakow, Henschen and others).

The ganglion cell is the trophic centre of the neuron, and, in the case of a direct motor neuron, of the muscle it supplies, but the condition of the cell is undeniably influenced by the degree of functional activity as well as by direct changes in the more peripheral parts of the neuron, the axis cylinder and its branches.<sup>3</sup>

This refinement in the minute anatomy of the nervous system is exceedingly important in connection with the various pathologic processes, and especially when it concerns the so-called primary system lesions of the brain and the spinal cord. These diseases are held by probably most writers as non-inflammatory, degenerative-atrophic processes in the physiologic tracts in which the motor and sensory neurons run in compact masses, *i. e.*, as primary diseases of the neurons, the nervous units.

The pathology of these primary system diseases may be truly said to be very obscure, although two etiologic conceptions have forced themselves forward into great prominence, namely, peculiar hereditary influences variously expressed, on the one hand, and certain forms of chronic intoxication on the other; most important among the latter is the post-syphilitic intoxication. Now it appears that either, or both together, of these factors, as well as other unknown causes, may seem to attack the neuron tracts in any part of their lengthy course, but as the integrity of the ganglion cell is so essential to the rest of the neuron, it follows that when this trophic centre is directly primarily involved, then the entire neuron quickly degenerates.



The histologic character and the topographic distribution of the typical neuron tract diseases certainly point to some such pathogenesis as this, inasmuch as the degenerative atrophy of the nerve elements, the neurons, in definite paths (mapping out with precision the Wallerian tracts of secondary degeneration and Flechsig's developmental tracts) is undoubtedly primary and the insignificant real changes of the stroma secondary. Actual inflammatory and coarse vascular lesions, on the other hand, are always diffuse and pay no attention to the imaginary anatomic limits of the various physiologic systems within their reach.

Among the few typical primary system diseases, amyotrophic lateral sclerosis with bulbar paralysis may be mentioned as illustrating exactly the pathologic and the anatomic considerations hinted at. It is a primary, non-inflammatory disease of both the direct and the indirect motor neuron—the entire motor tract. Surely a more subtle agent is at work here than in inflammations for instance, as it selects with unerring precision the scattered ganglion cells of various individual motor neurons, and consequently this disease has been held as quite strictly *sui generis* ever since Charcot first described it.

While amyotrophic lateral sclerosis is a disease of the two separate motor systems, and consequently in one way a combined system disease, yet the so called combined system diseases proper are those in which there are primary changes of the same cause and kind in sensory as well as motor tracts. The instances of combined system diseases described heretofore have almost all been cases in which but one of the two motor neuron systems, together with the sensory tracts, have been involved at the same time, the combined system disease *par* preference being primary degeneration in the posterior and the lateral columns.

A hurried review of the literature will make this statement clear.

Since the researches of Friedreich, Westphal, Kahler and Pick, and Strümpell placed the combined system lesions upon a firm clinical and anatomic basis, a large number of instances have been described.

Ormerod,<sup>4</sup> in a critical digest on the combination of posterior and lateral sclerosis, considered twenty cases.

<sup>4</sup> *Brain*, April, 1885.

Grasset,<sup>6</sup> in 1886, grouped together thirty-three cases which, viewed mainly from the clinical side, he termed instances of *tabes combinè* or *sclerose postero-laterale de la moëlle*. Dana<sup>7</sup> collected forty-six cases under the name of progressive spastic paraplegia or combined fascicular sclerosis. The most recent discussion of the subject is by Lenmalm<sup>8</sup> in an article in which he studies about 100 cases with post-mortems from the literature and twelve personal instances (he includes in this number cases of so-called diffuse combined sclerosis as well).

Considering for a moment the topographic distribution of the combined system lesions, then the following combinations are represented in the literature: combined system disease of the posterior columns and the pyramidal tracts;<sup>9</sup> the posterior columns and the cerebellar tracts;<sup>10</sup> the posterior columns, pyramidal and cerebellar tracts;<sup>11</sup> the posterior columns and the anterior gray horns;<sup>12</sup> the pyramidal and the cerebellar tracts;<sup>13</sup> the pyramidal and cerebellar tracts, anterior horns and posterior columns;<sup>14</sup> the pyramidal tracts, anterior horns and the posterior columns.<sup>15</sup>

<sup>6</sup> *Arch. de Neurologie*, xi., 1886, p. 156.

<sup>7</sup> *Medical Record*, xxxii., 1887, p. 1.

<sup>8</sup> *Hygiea*, lvi., 1894.

<sup>9</sup> Strümpell, *Arch. f. Psych.*, 1880, xi.; Erlicki and Rybalkin, *Arch. f. Psych.*, 1886, xvii. Dana, *Medical Record*, ii. 1887, p. 10. Oppenheim, *Neurol. Centralblatt*, 1888, p. 617.

<sup>10</sup> Francotte, *Arch. f. Neurol.*, 1890, xix.

<sup>11</sup> Friedreich, *Virchows Archiv*, Bd. 26, 27 and 70. Kahler and Pick, *Archiv. f. Psych.*, 1878, viii. Westphal, *Arch. f. Psych.*, 1878, viii. Strümpell, *loc. cit.* Smith, *Boston Med. and Surg. Jour.*, v. 113, 1885. Babinski and Charrin, 1886. Clarke, *Brain*, xiii., p. 356. Ruti-meyer, *Virch. Archiv*, Bd. 91 and Bd. 110. Lenmalm, *Hygiea*, lvi., 1894, p. 251. Schmaus, Neumann and Auscher are quoted by Lenmalm, *loc. cit.*, as describing this combination in probable syphilitics. Dejerine and Schmaus, *Semaine Med.*, 1894, p. 321.

<sup>12</sup> Blocq and Londe, *Anal. Pathol. de la moëlle epinière*, and Charcot and Marie, *Rev. de Med.*, 1886. Leyden, *Rückenmarks pr.*, ii. Bd., ii. Abth., p. 423. Marinesco, *Semaine Med.*, 1894. Dinkler, *Deutsche Zeitsch. f. Nervenheilk.* Bd. iv., 1893.

<sup>13</sup> Strümpell, *Arch. f. Psych.*, 1886, xvii. Münzer, *Wiener Kl. Wochenschrift*, 1892 (in this case Gower's tract was also involved). Min-kowski, *Deutsche Arch. f. Kl. Medicin.*, 1884, p. 433.

<sup>14</sup> Mayer, *Lehr. die comb. system. Erkr. der Rückenmarksstränge*, Wein. u. Leipsig., 1894. Lenmalm, *Hygiea*, B. lvi., 1894, p. 256. Leyden, *loc. cit.*, p. 441.

<sup>15</sup> Charcot and Marie, *Arch. de Neurologie*, x., 1885, p. 1. Leyden, *loc. cit.* Moeli, *Arch. f. Psych.*, x. 1880, p. 718. Rovighi and Melottie, *Riv. speriment. di Frenia et di Med. Cg*, 1888, xiv., p. 315; *Ext. in Neur. Centralbl.*, viii., p. 177.

NOTE.—This list is not by any means complete as far as the number of cases are concerned. Furthermore, other combinations are also possibly described, but their title as primary systematic changes might be seriously questioned.

Looked at in this way the combined system diseases form a heterogeneous collection containing very likely myelitic and other cases that might seem to justify Leyden's<sup>15</sup> remarks that all so called combined system diseases are myelitic in their nature, except Friedreich's disease, because their symptoms are so indefinite and because their lesions do not respect the limits of the physiologic tracts. Lennmalm<sup>16</sup> shows, however, that these cases are, to a certain extent, susceptible of an etiologic division, while the clinical manifestations and the morbid anatomy might not allow any differentiation; in this way more order and clearness is reached.

Thus the evident hereditary nature of so many of the cases of lesions in the lateral and the posterior columns places these in classes of their own, as witness Friedreich's hereditary ataxia and Strümpell's<sup>17</sup> hereditary spastic spinal paralysis.

The distinct toxic etiology of the lateral and posterior tract degenerations shown by Juczek<sup>18</sup> to follow poisoning with decomposed corn (pellagra) makes another member in an etiologic division.

A very large number of the remaining cases appear to be anomalous forms of tabes dorsalis, the tabetic symptoms and lesions being the predominating, but accompanied with changes in the lateral columns, or in the anterior horns.<sup>19</sup> If the prevailing syphilitic toxine theory of locomotor ataxia be accepted then it follows that the tabic group of combined lesions also forms a separate one from an etiologic point of view.<sup>20</sup> In a few of the cases of combined system disease in syphilitics the localization in the motor tracts has been predominating,<sup>21</sup> and it seems that the syphilitic group of combined

<sup>15</sup> *Zeitschrift für Kl. Medicin*, xxi., 1892.

<sup>16</sup> *Loc. cit.*

<sup>17</sup> *Deutsche Zeitschrift für Nervenheilkunde*, Bd. iv., 1893, p. 173.

<sup>18</sup> *Klinisch und Anatomische Studien über die Pellagra*, Berlin, 1893.

<sup>19</sup> Dinkler quotes several such cases of changes in the anterior horns (*loc. cit.*). Collins has a clinical case of tabes and progressive muscular atrophy in a syphilitic (*JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1894, p. 90.)

<sup>20</sup> Should Obersteiner's demonstration (*Centralblatt für Path. u. Path. Anat.*, B. v., 1894, p. 768) that the tabic spinal cord lesions are secondary and depend on a syphilitic meningitis, causing contraction of the pia, vascular sclerosis, and compression of the posterior roots, prove true in all cases, then it is likely that these cases of combined lesions would have to be explained in some other way than as primary degenerations.

<sup>21</sup> See as examples the cases of Leyden and Mayer (*loc. cit.*).

lesions may include almost all the topographic combinations possible, although the preference of syphilitic primary disease for the posterior, sensory tracts will remain a peculiar feature. It is easily seen that in complicated individual instances of syphilitic degeneration it may become difficult to classify the changes whether as primary or secondary, diffuse or systematic, or as mixed.

Then there remain some cases without any known clue to an etiologic grouping. Nearly all such cases have been described as primary degenerations in the pyramidal tracts and in the posterior columns, the clinical picture being an easily recalled combination with elimination of varying degrees of the characteristic symptoms of lateral and posterior sclerosis; as examples may be mentioned the instances described by Pierret,<sup>22</sup> Strümpell,<sup>23</sup> Babinski,<sup>24</sup> Lenmalm,<sup>25</sup> and others, in all of which the microscopic examination showed system changes in the posterior columns, the pyramidal, and occasionally in the cerebellar tracts, including often Clarke's columns, the anterior horns being normal, while the cerebral motor ganglion cells have usually not been studied, and the symptoms during life corresponded with reasonable precision to the lesions demonstrated.

Finally is reached an insignificant number of instances in which the lesions involve both the motor neuron-system and the sensory tracts, and it is to this combination that special attention is directed at this time.

First may be mentioned a few instances of this comprehensive union of changes in undoubted or supposed syphilities:

Leyden<sup>26</sup> found in a syphilitic with paralysis of the extremities, the tongue and the head, with some muscular atrophy, degenerations in the lateral, anterior and posterior (slight) columns, in the cells of the anterior horns, in the hypoglossus nuclei, and in the anterior nerve roots.

Mayer<sup>27</sup> has a case of tabes in a syphilitic plus spastic spinal paralysis and dysarthria; post-mortem he found

<sup>22</sup> *Arch. de Physiolog.*, iv., 1871-72, p. 367.

<sup>23</sup> *Arch. f. Psych.*, xl., 1881, p. 32 and p. 55.

<sup>24</sup> *Neurologische Centralblatt*, v., 1886, p. 140.

<sup>25</sup> *Loc. Cit.*, p. 251.

<sup>26</sup> *Klinik. der Rückenmarkskrankheiten*, Bd. ii., Abth. ii., 1876.

<sup>27</sup> *Loc. Cit.*

systematic lesions in the pyramidal, cerebellar and posterior columns, atrophy of the cells in the anterior horns, in Clarke's columns, in the hypoglossus, facial and sixth nuclei and of the posterior nerve roots.

Lennois and Lemoine<sup>28</sup> describe a case of atypic amyotrophic lateral sclerosis with optic atrophy and lancinating pains in a woman, 28 years old, without any history of syphilis. The changes were: Degeneration of the pyramidal and cerebellar tracts, of the posterior columns, of the cells in both anterior and posterior horns, and chronic changes in the medulla and pons.

Lenmalm<sup>29</sup> has an instance of tabes without any cerebral symptoms, combined with paralysis and some muscular atrophy. The changes were: Symmetric lesions of pyramidal, cerebellar and part of the posterior columns, with atrophy of the cells in the anterior horns and in Clarke's columns.

These instances may serve, then, as examples of a combination of system lesions in both the motor segments and the sensory paths with a more or less distinct syphilitic etiology. The resemblance of the clinical and anatomical features in two of these cases to amyotrophic lateral sclerosis recalls the typical cases of this disease with optic atrophy observed clinically by Suckling<sup>30</sup> and Peltesso<sup>31</sup> in syphilitics.

Then there are some instances of typical amyotrophic lateral sclerosis with system lesions in the posterior columns of slight extent, no symptoms pointing to their existence having been observed during life.

Charcot and Marie<sup>32</sup> found slight degeneration in Goll's columns in the two first cases of amyotrophic lateral sclerosis, in which degeneration was demonstrated in the ganglion cells of the cortex of the motor region, but sensory symptoms were not observed.

Macli<sup>33</sup> has a case of symptomless degeneration in Burdach's columns in amyotrophic lateral sclerosis. Rovighi and Melotti<sup>34</sup> describe in extenso a typical case

<sup>28</sup> *Arch. de Med. Exp.*, 1894, p. 443.

<sup>29</sup> *Loc. Cit.*

<sup>30</sup> *British Med. Journal*, 1882, vol. ii., p. 1,152; quoted by Lenmalm, *Loc. Cit.*

<sup>31</sup> *Centralblatt für Prakt. Augenheilkunde*, 1886, p. 6; quoted by Lenmalm, *Loc. Cit.*

<sup>32</sup> *Arch. de Neurolg.*, x, 1885, p. 1.

<sup>33</sup> *Arch. für Psych.*, x., 1880, p. 718.

<sup>34</sup> *Riv. Speriment di Fenetria e di Medicina Legale*, 1888, xiv., p. 315; Extr. in *Neur. Centralbl.*, viii., p. 177.

of amyotrophic lateral sclerosis in a 27 year old man, the clinical course running from October, 1881, when atrophy and weakness in the hands and forearms appeared, bulbar symptoms coming on in November, 1882, the picture being complete in October, 1883, to January, 1886, without any disturbance of sensation or sphincter function. There was atrophy of the nuclei in the medulla, of the ganglion cells in the anterior horns, total degeneration of the crossed pyramidal tracts, with some degeneration in Goll's columns and atrophy of both anterior and posterior nerve roots, the cells in Clarke's columns being normal.

Oppenheim<sup>35</sup> has a case with sharp, systematic degeneration of Burdach's columns ceasing precisely at the septum intermedium, the cells in Clarke's columns being atrophic, and of this definite lesion he states positively there were no symptoms.

Marie,<sup>36</sup> in his description of the morbid anatomy of amyotrophic lateral sclerosis, states that the columns of Goll often show some slight degeneration, but that the characteristic "corps granuleux" are absent. No cause for this degeneration is known, but he hints at its possibly being due to lesion of the cells in the posterior horns.

Leyden<sup>37</sup> mentions a case of amyotrophic lateral sclerosis with slight prodromal symptoms of a tabic nature, to wit, anæsthesia of the plantar surfaces of the feet, and post-mortem slight degeneration in the posterior columns was found in addition to the usual motor tract lesions.

Oppenheim<sup>38</sup> has an instance of rapidly fatal amyotrophic lateral sclerosis. In addition to the typical symptoms there was hyperæsthesia in the left thoracic region and diminished pain and temperature sensibility in the right leg and foot. The characteristic lesions in the motor tract were demonstrated, and also a circumscribed lesion of the left posterior horn and the left posterior nerve root in the dorsal region corresponding to the origin of the second and third dorsal spinal nerves—a unique example of amyotrophic lateral sclerosis and a diffuse sensory tract lesion.

Finally, Raymond's<sup>39</sup> case may be brought in. A

<sup>35</sup> *Arch. f. Psych.*, xxiv., p. 758; *Neur. Centrbl.*, 1888, No. 6.

<sup>36</sup> *Traite de Médecine*, 1894, Tome vi., p. 340.

<sup>37</sup> Quoted by Omerod, *Loc. Cit.*

<sup>38</sup> *Arch. f. Psych.*, xxiv., p. 758.

<sup>39</sup> *Arch. de Phys.*, 1892, x.

woman, 78 years old, who had had pain in her legs since her fortieth year, suffered during the last years of her life with spastic paralysis, increased reflexes, wasting of many muscles without electric change, and post-mortem there was found in addition to arterio sclerosis rather irregular degeneration of the posterior columns, of the pyramidal and cerebellar tracts, with absence of many cells in the anterior horns and in Clarke's columns. This case is justly regarded by Lennmalm as an example of an arterio-sclerotic diffuse lesion, and is introduced in this manner simply for the sake of completeness and comparison.

These, then, are apparently all the cases from the long and complicated list of combined system lesions in the spinal cord that present at the same time primary changes in both motor neuron systems and in the sensory tracts. Much commentary is not necessary, because it will be observed that the syphilitic cases present a peculiar and varying mixture of the symptoms of tabes and of chronic spinal muscular atrophy, and the cases of amyotrophic lateral sclerosis accompanied with system changes in the posterior columns are disappointing in so far as corresponding sensory symptoms were absent (or not observed). In Leyden's single case only was there any disturbance of sensation—anæsthesia of the soles of the feet—and this was a prodromal symptom merely.

In other words, a typical case of amyotrophic lateral sclerosis, with bulbar paralysis and with the sensory symptoms of a genuine posterior sclerosis, does not seem as yet recorded. This condition is quite closely approached, however, by some of the instances of primary combined syphilitic lesions that have been mentioned.

Should this state of affairs be interpreted as meaning that the cause of amyotrophic lateral sclerosis and bulbar paralysis has such affinity for the motor neurons as to always leave the sensory uninvolved? In fact, the marked affinity of the syphilitic poison for the sensory neurons and the almost exclusive involvement of the indirect motor neurons in the hereditary forms of combined sclerosis point to some such explanation as suggested in the above question.

From this consideration it becomes plain that the following case of primary system disease of both motor neurons and of the direct sensory neurons presenting the clinical picture of amyotrophic lateral sclerosis with bulbar paralysis, in the course of which marked sensory

disturbances develop, becomes an important addition to the literature of the system diseases.

The case was observed clinically in Prof. Henschen's wards of the Upsala Academic Hospital; the organs of the nervous system were examined and studied by me under Prof. Henschen's supervision in his laboratory. I am deeply indebted to Prof. Henschen for placing the clinical records, the organs, and his laboratory at my disposition, and I thank him sincerely for his kindness, aid and advice.

#### THE CLINICAL HISTORY.

J. S., born 1825, farm laborer, admitted Aug. 7, 1890. His parents died fifty years ago of unknown causes; two sisters live and are well; one sister died two years ago. As far as he knows, no instance of a disease like his ever occurred in the family, which has always been regarded as strong and healthy. He is married and father of three sons, all of whom are soldiers (consequently well built, healthy men). He has always lived in the country, in good hygienic surroundings. He has had good health until this illness came on. He is ignorant of having had any of the diseases of childhood. While young he had chills and fever, and in 1857 an attack of small-pox; ten years ago his chest was crushed in and a rib was broken, but perfect health was soon regained. During the fifteen years after 1860 he was a bridge-tender, and was often subject to mental worry and sudden, momentary physical over-exertion; otherwise he has always been a farm laborer.

He has never used alcohol to excess, and denies all venereal diseases. In 1875 a brief attack of unconsciousness came over him, during which he fell to the floor; spasms, palsy or aphasia were not noticed; in a few days he was quite well again. Subsequently similar spells appeared from time to time; an attack in 1885 was characterized by twisting of the head down and to the right; the last attack occurred in 1887, and as it was ushered in by noises in the ears and flashes of light before the eyes, he had time to lie down before unconsciousness supervened. These attacks were all of very short duration, and, with the single exception of the one in 1885, consisted in momentary losses of consciousness, during which he would fall.

The actual commencement of his illness he traces



back to July, 1887; he can give no cause for the gradual weakness and increasing trouble in walking which then began. Soon after this time the left foot became stiff and painful, the pain extending into the thigh; he was treated in this hospital from January 20 to February 19, 1888, the diagnosis being chronic muscular rheumatism. The following is extracted from the records of this visit: "Muscular, healthy looking man; bodily functions normal; passive motion in the left foot is painful, and walking causes pain in the left calf, while the foot drags behind some, touching the floor very lightly. The left foot is a little swollen, but sensation is normal." Now (1890) the patient claims that some, though slight, weakness was present in his right leg during and even before this short visit to the hospital. In the spring of 1888 he gave up his work, principally on account of the trouble with walking; in the fall of the same year he had to use support in walking; at this time shooting pains came into the lower extremities, and the back was stiff and painful; the upper limbs also became weak; frequently they would "sleep;" this was noticed first in the left arm; the hands grew thin, the fingers stiff, so that he could not dress and undress himself unaided. Speech also became troubled; he could express himself if given time, and he did not forget the names of persons or objects, but it was hard to pronounce clearly, and he had to talk slowly. Nor was this all, for chewing and swallowing were also disturbed, and he could not prevent mucus from dribbling from his lips or trickling into the pharynx. Sensation, defecation and urination were unchanged. At New Years, 1889, he fell and hurt his chest some.

In March, 1889, he was readmitted to the hospital, remaining until June 8, the same year, the diagnosis now reading amyotrophic lateral sclerosis. The following is an extract from the records of this visit: "Shooting pains in the legs and in the back; it is hard for him to chew and to spit; there is no aphasia; vision good, smell and taste seem a little dulled; there is a slight paresis in the left facial nerve; the movements of the tongue are limited, the point deviating a little to the left, swallowing is labored, speech indistinct, the voice hoarse and monotonous. Sensibility everywhere normal. The hands are thin, and the movements of the fingers, hands and feet weak and slow; the left leg is weaker than the right; both feet drag behind when walking, and the legs

tremble much as soon as he tries to hurry. Reaction of degeneration is present in the adduct. digit min., abd. pollicis brev., peroneus long. dext., and in both the ext. com. long., while in the adduct. pollicis and abd. hallucis the excitability is lessened. The patellar reflexes are exaggerated.

Note May 29, 1889: "The forearms are flat and the interossei wasted, leaving depressions. The bulbar symptoms are prominent."

When he left the hospital (June 8, 1889) he walked by the aid of two canes. At the end of 1889 he could not move from chair to chair unaided. During this time he thinks that sensation also became lessened, first in the limbs of the right side, then in the left. In the early part of 1890 he often fell in trying to move from place to place; he became more and more helpless, and was brought back to the hospital August 7, 1890.

#### EXAMINATION, SEPTEMBER, 1890.

The patient is bedridden, and cannot unaided change his position.

Emaciation is marked, the hands especially being thin. No œdema, no decubitus. Sleep and appetite good; urine normal. Obstinate constipation. He cannot cough or spit out saliva, which accumulates in pharynx. No fever. Perception, judgment and memory seem normal. He is emotional, passing easily from smiles to tears. There is no aphasia, and, as he cannot write or read writing, he readily puts together words by means of loose letters.

#### *The Cranial Nerves.*

I.—No hallucinations. In the left nostril the smell seems a little lessened.

II. Vision good; no hemianopia.

III., IV. and VI.—The movements of the eyes are normal; the pupils equal and active; no ptosis, nystagmus or strabismus.

V.—Sensibility in all forms normal. Marked increase in the salivary secretion. Mastication is slow and feeble; the buccal mucous membrane often falls in between the teeth. He eats only soft bread and finely chopped food.

VII.—He wrinkles forehead well, perhaps a little livelier on the right side. The right eyelids are closed tighter than the left. The lips move stiffly, and often

allow food to fall out when he is eating. The left palatal arch hangs lower than the right; uvula does not deviate. Emotional movements in the face are active.

VIII.—Watch heard at 15 cm.; no hallucinations.

IX. (V.)—Taste is diminished; salt, sweet and sour are not definitely distinguished between along the left half of the tongue, particularly at the tip. He says he tastes his food less than formerly. No hallucinations. Deglutition is laborious, semi-solid articles giving less trouble than liquids, which run into larynx and cause cough.

X.—Respiration and pulse normal. Sensation in larynx normal.

XI.—Voice monotonous. Adduction of cords less prompt than normal. Movements in sterno-cleido-mastoid and trapezii muscles feeble.

XII.—The tongue is flat and thin; it can be put out only as far as the margin of the lips, and the tip points to the left; on the back and along the margins are fibrillary twitchings. It cannot be pressed firmly against the roof of the mouth. Efforts to pull it out are vigorously resisted.

Speech.—A monotonous sound is produced, in which there is but little accentuation. "Yes" and "no" (Swedish) are tolerably distinctly pronounced. Of the letters, "h" (Swedish) is best pronounced; the vowels come next; and of the consonants "f," "m," and "v" are clearest.

### *The Peripheral Nerves.*

Sensation.—Examination on this point is somewhat difficult on account of the dysarthria. The following nearly completely anæsthetic districts are, however, easily demonstrable:

I.—The district supplied by the right ulnar and right internal cutaneous nerves. The included part of the hand and fingers and of the lower third of the forearm are about completely anæsthetic.

II.—The left little and ring fingers, the ulnar half of the hand and of the forearm up to 18 cm. above the styloid process.

III.—The right leg up to 7 cm. above the patella, inclusive of dorsum of foot.

IV.—The left leg up to 3 cm. above the patella, including the foot and the toes, except the plantar surfaces.

In these areas tactile sensation is lost. The sense of pain is dulled, but in the anæsthetic part of the right hand and forearm it is lost. Temperature sensibility is also diminished, and most in the right upper extremity. It is evident that the anæsthetic areas are daily becoming less in extent. Elsewhere sensation is normal, and the points of the æsthesiometer are felt as two at the usual distances, but on the legs and forearms (anæsthetic districts), only when 20 to 25 cm. apart.

*Motility.*—The head is moved freely. The trunk is stiff; bending it is impossible; cannot voluntarily change his position. The pectoral muscles are atrophic. In the shoulder joints motion is natural; at the elbow extension is limited, and requires much passive force; supination and pronation are restricted; motion in the fingers is diminished, and occurs slowly and fumblingly—cannot button his shirt. He cannot straighten all his fingers; passively all can be made straight. The muscles in the arms and forearms are thin and flaccid, except that there is some contraction in the biceps. The thenar and hypothenar eminences are flattened, and on the dorsum the intervals between the metacarpals are deep and marked.

The legs cannot be lifted from the bed. He can only partly flex hip and knee joints; ab- and adduction are also much restricted. The passive range of motion is normal. The feet are thin, and the leg muscles soft.

*Electric Examination.*—The reaction of degeneration is present in the interossei, thenar and hypothenar eminences, the right tibialis anticus, and peroneus longus, in the left gastrocnemius and adductor hallucis. There is diminished excitability in the left facialis district.

*Reflexes.*—No maxillary reflex. Biceps reflex increased. Triceps reflex absent on the right side, present on the left. Exaggeration of both patellar reflexes. Left ankle clonus. Plantar reflexes strong. Scrotal reflex normal. Abdominal reflex cannot be produced.

Mechanic irritability is increased in the muscles of both forearms, in triceps and biceps muscles, and in the anterior muscles of the left leg.

*Trophic Changes.*—In addition to the muscular atrophy there is nothing to remark.

*Vegetative Organs.*—Nothing abnormal to note, except bilateral mucous rales in lungs, posteriorly and laterally.

[From the records of subsequent complete examina-

tions only the very salient and new features are here introduced.]

October 17, 1890.—Anæsthetic areas gradually fading away; and October 24, 1890, are now absent in the legs. Right ankle clonus now present; bilateral triceps reflex.

November 11, 1890.—Anæsthetic areas now limited to little fingers; ankle clonus absent.

November 28, 1890.—Wind passes involuntarily. Anæsthesia has disappeared.

February, 1891.—Complains of pain in right forearm along ulnar nerve and thence into axilla. Efforts at coughing result simply in a prolonged expiration, with gurgling in the throat. Dysarthria aggravated.

Ophthalmoscopic examination negative. Æsthesiometer results are now like the normal everywhere.

May, 1891. Pain in right arm worse. Right vocal cord paretic. Contractures in biceps and forearm flexors. A light touch is not felt on the skin of the trunk and the extremities; neither can he distinguish definitely between head and point of a pin. Thermo-sensibility seems reduced inasmuch as he cannot distinguish any smaller differences in temperature than  $+ 2^{\circ}$  c.

He is receiving massage with apparent benefit as far as the muscular wasting is concerned.

November, 1891. More and more helpless. Pain in the right arm persists, and there is now aching in the left ulnar district, in the right foot and in the back. He does not sleep well on this account.

Feces now pass involuntarily, but he is master of his bladder.

The visual fields are concentrically limited; eye-grounds normal; the movements of the eyes jerky and convergence limited. Cannot raise his head from the pillow. Left half of tongue atrophic.

Tactile sensation diminished in the hands, forearms and feet; and when pricked quite sharply with a pin he feels no pain; a test-tube filled with boiling water causes pain only after a few seconds. Ulnar districts show further loss of thermo-sensibility, otherwise this is as in May. Muscular sense good.

He cannot now lift his arms from the bed, and the interossei are completely absent; tremors are now and then visible in the forearms. Ankle clonus again present. Reaction of degeneration present in muscles of hands, legs and feet.

February, 1892. Emaciation and cachexia marked. Is fed with tube. Both urine and *féces* now pass involuntarily. The urine contains albumin. There is redness over all prominences. He understands what is said to him, but anarthria is extreme. Concentric limitation of visual fields; movements of eyes slow.

Temperature and pain sensibility in the face is diminished; does not recognize a difference of  $+7^{\circ}$  c. on forehead  $+6^{\circ}$  c. on cheeks.

The masseters are atrophic, a bite on the finger is hardly felt. The uvula deviates to the left. Cannot distinguish between sweet, sour and salt. Sterno-cleido-mastoids and trapezii atrophic.

Peripheral nerves; tactile sense as before; feels a pressure of 50 grams and distinguishes differences of 50 gr. Thermo-sensibility further diminished; with one thermometer constantly at  $+28^{\circ}$  c., a difference of  $+9$  to  $+15$  degrees is the first to be felt on hands and arms,  $+14^{\circ}$  c. on chest,  $+9$   $+12$  on legs. On arms  $-10^{\circ}$  c. is pointed out as cold, on legs  $-10$  to  $15$ , above  $-5$  is not recognized as cold anywhere. This shows sensibility to cold to be lessened also. Sensibility to pain is symmetrically diminished; with Björnström's algometer pain is indicated on arms at 7 to 8 kgm., chest 7, legs 8, 9. Pain is gradually and slowly produced by bringing the skin in contact with boiling water. An electric current from 60 elements does not cause pain. The points of the æsthesiometer are felt as two at a distance of 70-80 mm. on the arms; 60-65 on the volar surfaces, and 70 on the back of the hands; 70 on the chest, and 70-80 on the legs.

Contractures at right angles in the elbow joints. Main en griffe.

March 13, 1892. Alb. 4-6 per cent. in the urine. Extensive sacral decubitus. A firm pinch of the skin of the face is felt as a touch; on the right side after 1 second; on the left side after 1.5-2 seconds; as pain, right side, after 6-8 seconds; left side, 7-8 seconds.

Peripherally a firm pinch is recognized as a touch after 1-3 seconds, as pain after 3-8 seconds, the retardation most marked in the legs.

Temperature-sensibility as in February, but more diminished.

March 22, 1892. Moribund. Temperature,  $38.5$ ; pulse, 160; respiration, 40. Occasional trismus. Pupils react to light. Active patellar reflexes, do. plantar.

Died March 23, 1892.

## THE MICROSCOPIC EXAMINATION.

*Post Mortem.*—This was limited to removing the brain, spinal cord, tongue, pharynx, larynx, and the cranial and a few peripheral nerves. To the naked eye the cerebral hemispheres and their coverings were normal; there was no atrophy of any convolutions visible, but the pyramids of the medulla were flatter than usual and the bulbar nerves noticeably thin and yellow.

The spinal dura was normal, but the pia seemed thickened some; the vessels did not show any macroscopic changes. The spinal cord was small, atrophic, but of uniform consistence and natural color and contour. On the cross sections the posterior part of the lateral columns presented bilaterally symmetric areas of gray color. There were no cavity formations or softening.

The tongue seemed small, thin and flat; otherwise normal appearance in tongue, pharynx and larynx.

The above observations are corroborated by the appearances of the hardened specimens.

*Technique.*—Small pieces from the cortex of all parts of the central convolutions and segments from the various levels of the spinal cord were hardened in alcohol, imbedded in paraffin. The sections fixed on the slide with oil of cloves-collodion, the paraffin extracted with xylol, and the xylol with alcohol; staining in a .5 per cent. aqueous methylen blue solution was now done under gentle heat until steam arose, decolorization in alcohol-anilin oil, cleaning in origanum oil and mounting in benzine-colophonium (Nissl's method).

The brain and cord were otherwise hardened in Müller's fluid and Weigert preparations made from all parts of the cord, the right central ganglia and pyramidal radiation. The medulla and pons were cut in an uninterrupted series, and about every tenth section stained. Nuclear stains were also employed. The cranial and peripheral nerves were examined by means of teased arsenic acid preparations. The spinal nerves and ganglia were examined in Weigert and nuclear stains. The tongue and other muscles were studied in specimens made after the ordinary histologic plans. Specimens from the nervous system were also stained after the Stroebe\* and Van Gieson\* methods.

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\* *Centralblatt für Path. u. Path. Anat.*, 1893 and 1894.

*The Brain.*

1. Cortex of Central Convolutions.—Methyl blue sections show, in comparison with normal specimens made in the same way, a probable diminution in the number of large pyramidal cells. It is also noticeable that it is difficult to bring out the axis cylinders and protoplasmic processes, even though the cell body and nucleus may stain well and appear normal. There are no gross changes. Occasional pyramidal and ganglion cells are found which appear decidedly changed either in having lost their prolongation or nuclei, or in staining very diffusely and presenting very abnormal shapes. A few granular, rounded or irregular, masses are also found in the region of the large pyramidal cells. (Fig. 1.). As far as observed such changes are best marked in the central, then in the lower, and least often in the upper parts of the anterior and posterior central convolutions of the two sides.

In the Weigert preparations no changes are recognized and any atrophy of the fibræ propriæ, or association fibres, cannot be demonstrated.

2. Pyramidal Radiation.—A large number of sections were made of the right pyramidal radiation, but degenerated fibres could not with certainty be made out.

3. Right Central Ganglia.—There is a distinct area of lighter color than elsewhere in the middle third of the internal capsule, *i. e.*, opposite the globus pallidus. This slight degeneration is uniform throughout this entire part of the capsule. Otherwise the right central ganglia are normal; the left was not examined. The optic tract is normal.

4. Crura Cerebri.—The only change observed is a rather faint but uniform degeneration in the part of the crura occupied by the pyramidal tract.

5. Pons.—(a) Pyramidal Tracts: Distinct, uniform, though slight degeneration in the scattered bundles, but marked in smaller masses of fibres.

(b) *Formatio reticularis*, the fillet, the posterior longitudinal bundle, and the crura cerebelli ad pontem are all normal.

(c) Abducens, trochlearis, and oculo-motorius nuclei are also normal.

(d) Trigemini.—The motor nucleus contains a few atrophic cells and there is some sclerosis of the ground substance. The motor roots contain some degenerated fibres. The sensory nucleus cannot be shown to contain



markedly changed cells, but the sensory nerve roots have degenerated fibres. Ascending and descending trigeminal roots seem unchanged.

6. Medulla.—(a) Pyramids: Uniform, moderate thinning of the fibres is presented in both these columns and is apparently more pronounced than in the pons or the internal capsule. There is no marked nucleus increase, a few large, round cells are present, and the vessel walls seem thick.

(b) Facial Nucleus.—Many excessively pigmented cells are found without prolongations; also, granular masses of pigment; many normal cells are present and the ground substance does not seem so sclerotic as in other nuclei. No changes are found in the knee, but the roots are very thin.

(c) Nucleus viii. and roots are normal.

(d) Spinal accessory, vagus, and glosso-pharyngeus nuclei.—These present a considerable degree of atrophy with evidently changed cells and sclerosis; the nerve roots are thin. The solitary fasciculus seems normal. Nucleus ambiguus atrophic.

(e) Hypoglossus Nucleus.—There is marked atrophy of the cells in the principal nucleus. Roller's small-celled nucleus seems unchanged. The nerve roots are very thin, but degenerated fibres are not seen. The healthy roots would correspond to a few normal cells still remaining. There is marked sclerosis and degeneration of the nerve network in the nucleus and the white matter surrounding the latter upon its ventricular borders has disappeared entirely. (Fig. 2).

(f) Funiculi Graciles.—There is slight uniform atrophy in the nerve fibres corresponding in degree to the change in the Goll's columns. The cells in the nucleus funic. gracil. appear quite normal.

(g) Funiculi cuneati, restiform bodies, olivary bodies, nuclei arciformes, formatio reticularis, and lemniscus are all normal.

(h) Central Gray Matter.—There is some accumulation of round cells about the central canal in the distal end of the medulla and the gray matter in the floor of the fourth ventricle is thicker than normal.

6. Cerebellum.—Methyl blue specimens from the cortex of both hemispheres present plainly normal conditions in all respects.

#### *The Spinal Cord.*

1. The Pia.—The vessels in the pia have thick walls,

the arteries especially. The thickening is uniform, involves particularly the media in which the number of nuclei is increased, but these are also endarteritic changes, although the intima is always smooth and presents no thrombotic deposits. The thickening extends along the entire course of the posterior and the anterior spinal arteries. In the small vessels in the posterior longitudinal septum the walls may have a homogeneous, glassy appearance, staining diffusely red with eosin and acid fuchsin. The walls of the veins are also quite thick and often homogeneous. There is no perivascular round cell infiltration.

The pial meshes seems somewhat denser in their fibrillation than usual; distinct inflammatory changes are absent.

2. Anomalous Vein.—At this time may be described a vascular peculiarity in the distal dorsal region of the cord. Here a rather large, fusiform, thin-walled, venous channel cuts across the central commissure from the bottom of the anterior to the posterior longitudinal fissures, passing between Clarke's column and the central canal. The bulging is greatest in the commissure, while the veins in the fissures do not seem at all enlarged. This venous channel has thin walls of connective tissue, filled with blood; the cord tissue on all sides is entirely unchanged. Serial sections including the entire extent of the dilatation were made and its vertical diameter would be about 5 mm.

It may be dismissed from further consideration as an anomaly without pathologic significance in this case. (Fig. 5).

3. The Central Canal.—It is filled with small, round cells so that the lumen is often loosely occluded, the normal lining being entirely disarranged. This cell accumulation does not encroach upon the adjacent structures and shows no tendency to infiltration; it is largest in the dorsal region.

4. The White Matter.—The sections of the cord are considerably smaller in circumference than those of a normal cord from a man of the same age, prepared similarly.

The following changes in the cord substance have to be described: Systematic degeneration in the pyramidal tracts and in Goll's columns, atrophy of the ganglion cells in the anterior horns and in Clarke's columns.

(a) The Antero-lateral Columns.—Degeneration in the

lateral columns is present throughout the entire cord. In the lumbar region it has a triangular shape, the apex pointing medianwards, nearly touching the conular junction; the ventral limit is nearly on a line with the central canal, while the dorsal border touches the posterior horn, and the external border or base corresponds with the periphery of the cord. The degeneration is most intense in the central part of the area thus outlined, but a few normal fibres are present even here. In the dorsal region the degenerated area occupies relatively the same position; it is, perhaps, more marked and does not reach the peripheral margin as a narrow strip of healthy substance intervenes—the direct cerebellar tract. In the mid and upper dorsal regions the degeneration is a little bit smaller in extent on the right side, and simultaneously with this disproportion between the two sides comes a crescent shaped district of degeneration in the left anterior column along the longitudinal fissure. Throughout the cervical region the changed district occupies the same relative position, and can be followed on both sides up to the decussation without any perceptible change in size or shape. (Fig. 3).

As one follows the changes upward the crescentic degenerated area in the anterior column becomes broader, and a very faint atrophy can be made out along the mesial border in the right anterior column, but the area of degeneration in the right lateral column remains noticeably smaller than in the left. (Fig. 3). Otherwise the antero-lateral column is normal.

(b) *The Posterior Columns.*—Already in the lumbar enlargement is observed a thinning in the medullated nerve fibres along the mesial borders of Goll's columns. In the dorsal region this degeneration becomes broader and involves to a limited but nearly equal degree all parts of both these tracts, the change being furthest advanced along the mesial border, and, becoming better and better marked, as it is followed in about this shape up to the nucleus of the funiculus gracilis in the medulla. (Fig. 3). Burdach's columns seem normal throughout.

(c) *White Commissure.*—In many sections, especially from the cervical enlargement, there is observed an irregularly distributed but distinct degeneration in the fibres of the anterior white commissure.

(d) *Nerve Roots.*—In the anterior nerve roots there is nearly without exception some degeneration, and in many instances this may be very intense. The posterior

nerve roots are also changed, but not so much as the anterior, and in the lumbar region many posterior roots show no marked atrophy.

5. The Gray Matter.—(a) The Anterior Horns: These are small but symmetric, retaining their normal shape. In the Weigert sections their color is more pronounced yellow than usual.

The following summarizing description of the ganglion cells is based mainly on the appearances observed in sections stained with methylene blue. In general it may be said that the changes consist in an exceedingly great diminution in the number of the ganglion cells due, so to speak, to a progressive degenerative atrophy or necrotic process. The changes in the stroma are very slight in degree.

The largest number of normal ganglion cells are found in the lumbar region; in the higher levels of the cord the wasting is so great that in the majority of the sections but few cells are present and many of these are greatly altered. In the cervical enlargement the mesial groups are almost entirely absent and the majority of the cells in the lateral groups are also more or less altered, so that frequently there are sections in which the normal ganglion cells are altogether absent, or at least the exception. Where the intermedio-lateral tract is distinct its cell are also more or less changed, but their number remains considerable.

The altered cells assume all sorts of forms. Any classification of the changes is hardly possible, inasmuch as there seems to be no other basis for division than the morphologic. In the examples of apparently early changes the cell body appears shrunken, usually without any distinct nucleus, and staining deeply and diffusely, while the processes may remain fairly well marked; often such cells are surrounded by a small empty space. (Fig. 4.) Then there are a large number of oval or rounded forms, often with an even outline, but frequently showing irregularities as though the processes had been broken of; in such cells it is surprising to often observe a quite typical looking nucleus. Here the cytoplasm also stains diffusely either wholly or partly, so that at portions of the periphery there may be light or deep-blue granules or particles scattered about. Cellular forms, usually rounded in outline, composed of colored and colorless granules, irregularly arranged, without indication of nucleus and with thin and

imperfect prolongation, are also found. In many abnormal cells one part of the cell body may contain light brownish, fine pigment granules, and the remainder may be diffusely and deeply stained—nucleus and processes being more or less disturbed or completely absent. (Fig. 4). Around changed cells of all forms may at times be found empty spaces on all sides or only part of their circumference. While the cells above indicated are all smaller than the normal ganglion cells, yet their dimensions are quite considerable, and there remains to mention that there are also many oval, rounded, angular and irregular forms of a comparatively smaller, but varying size, that stain either deeply and diffusely, or present a granular appearance, the granules being scattered about either thickly or more thinly; these have no sign of nucleus, an even outline and no processes. Finally, there are loose granular masses of variable size and all possible forms, occasionally presenting small, pointed projections that probably are remnants of protoplasmic prolongations or axis cylinders, and also more minute nondescript clumps and masses; the smaller cellular remains usually lie imbedded in the stroma without any empty spaces about them. They stain deep red with eosin acid and fuchsin, etc. In the Weigert sections the changed cells present a yellow or brownish color with very frequent large pigment heaps of a deep brown color.

The ground substance is rather granular, and only seldom can it be said that any distinct fibrillation is present. There is no marked accumulation of round cells. The vessel walls are a little thickened, but there is no perivascular infiltration, and the lumina are patent. There are no recent hæmorrhages or tokens of ancient ones present. Only a few glia cells are demonstrable. There is considerable degeneration of the medullated network in the anterior horns, many atrophic fibres and also myelin detritus being present, but the change is not excessive or complete.

From the above summary it will be seen that it is possible to trace with reasonable ease the apparent changes in the ganglion cells from the earlier to the latter stages. Among the earlier changes may be mentioned a shrinking of the cell body, with a deep and diffuse stain of the cytoplasm, followed by gradual loss of prolongations, the formation of a more or less distinct vacuole around the cell, while the general appearance of

the nucleus may remain apparently unchanged, although in many cells it soon disappears. Then follows a gradual diminution in the size of the cell, marked changes in its forms, which generally has a tendency to become rounded, and at times the cytoplasm stains diffusely, and at other times colored and colorless granules are present. Finally, there remain irregular clumps and granular masses that evidently represent dead cells in the process of removal; these small remnants lie in the stroma, which surrounds them quite closely on all sides.

As to how much of the above-mentioned apparent changes may be artefacts cannot be stated, but certainly the gross changes in form and size are pathologic, and stages in the necrotic process that finally results in the complete disappearance of the cells.

Concerning the finer structure of the nucleus nothing can be said, because the tissues examined were not instantaneously killed and fixed; in general, the cells taken to be normal (lumbar region especially) corresponded fairly well to the histologically perfect specimens as they present the characteristic vesicular nucleus, the peculiar, colored granules and particles in the cell body, and well-developed prolongations.

(b) Posterior Horns.—In the posterior horns proper it was not possible to demonstrate any changes either in the cells, nuclei, or stroma, but in Clarke's columns the number of cells seems diminished in some of the sections from the distal dorsal region. The majority of the cells are normal, but changed cells are present in small numbers in the shape of rounded shrunken forms, granular or homogenous, and of angular and irregular small masses. Any marked changes in the stroma and in the nerve fibres in or about the columns does not exist.

*Spinal Nerves and Spinal Ganglia.*—The changes in the spinal nerve roots have been mentioned in connection with the spinal cord.

In the Weigert sections across the cauda equina markedly degenerated bundles are observed.

The spinal nerves and the ganglia were studied by means of longitudinal and cross sections; in several instances a complete series of longitudinal sections were made through a short segment of the nerve, the ganglion and the roots.

In such longitudinal sections degenerated nerve fibres could be followed into the ganglion from the spinal

cord and among the nerves leaving the peripheral pole of the ganglion. Occasional degenerated fibres could be followed into the mixed spinal nerves.

In the spinal ganglia (Fig. 6) there is found, in addition to the moderate degree of degeneration of the nerve fibres, a number of changed ganglion cells; there are excessively pigmented cells, a few shrunk forms, thick, yellow, scale-like cells without nuclei, and also a few pale cells evidently in process of disintegration, the cytoplasm being finely granular and vacuolated, while the cell capsules seem enlarged. The cells regarded as excessively pigmented are filled so as to hide the nucleus (if it be present) with yellowish brown or quite black granular pigment (similar appearances in Weigert, Ehrlich, Biondi, and eosin-hematoxylin stains). The shrunk cells have no processes extending out to the walls of the capsules. There is no evident or marked increase in the nuclei of the ganglion cell capsules, and no striking change in the interstitial tissue, which is perhaps increased some.

*Cranial and Peripheral Nerves.*—Teased osmic preparations were made of all the cranial, the ulnar, median, and radial nerves; also of the cauda and of the left sympathetic nerve of the neck.

Typical pictures of degeneration, as shown by the breaking up of the myelin into characteristic, smooth, large and small, globular and oval lumps, were found, in varying degree, in the following nerves: A few degenerated fibres in the third pair, in the right fourth and the left sixth nerves; in the motor part of the fifth pair, and the sensory portion of the left fifth nerve; also in the seventh, ninth, tenth, eleventh and twelfth pair of cranial nerves, and of those especially in the ninth and right twelfth nerves; further, in the cauda, in both ulnar nerves to a marked extent, and, to a moderate degree, in the right radial and median nerves.

In many places the broken-up myelin had been taken away, as shown by the presence of droplets among granular detritus in sheaths that were empty between the heaps of such remains.

Apparently empty sheaths and thick as well as thin varicose threads were present also; granular myelin tubes, broken across at frequent intervals, were also seen; varicose threads and granular myelin sheaths were also found in those nerves that did not show any typical degeneration, but did not seem so frequent as in the degenerating nerve bundles.

The osmium preparations do not demonstrate any interstitial changes.

*Left Cervical Sympathetic Nerve.*—In this are found many myelinic fibres, the seat of typical degeneration.

*Tongue, Laryngeal and Pharyngeal Muscles.*—The mucosa of the upper and under surfaces of the tongue is normal, but the submucous connective tissue is increased some, and shows considerable round cell infiltration. The muscular fibres seem thin, the transverse striation indistinct, and the nuclei frequent; there are fibres which are narrower in one part of the longitudinal section than in others. Distinct fatty degeneration is not observed. The amount of connective tissue between the muscles and in the perimysium is increased, and in the deeper parts of the tongue much fat is present between the fibre bundles. The muscles of the larynx and pharynx show much atrophy; fibrous tissue replaces areas of muscular structure; small and narrow strands of muscle fibres are found here and there, and in such the transverse striation is indistinct.

#### CLINICAL SUMMARY.

A farm laborer, born in 1828, with negative family and personal history, has a few brief, sudden attacks of unconsciousness between 1875 and 1887; only one attack was marked by spasm (twisting of the head downward and to the right). In July, 1887, when he was sixty-two years old, weakness and trouble in walking appeared; pain, stiffness, and swelling in the left foot developed in addition, and was diagnosed as chronic muscular rheumatism in the early months of 1888. Soon the arms and hands became weak and thin; disturbance in deglutition, chewing and articulation also appear. In March, 1889, the diagnosis of amyotrophic lateral sclerosis is made, based upon spastic paresis in the extremities, with increased reflexes, muscular atrophy (hand muscles especially), and the reaction of degeneration, together with quite prominent bulbar symptoms, to wit: Dysarthria, difficult deglutition, paresis of the tongue and the muscles of mastication, etc. At the end of 1889 locomotion becomes impossible. General helplessness increases, so that by September, 1890, he is rendered permanently bedridden. The spinal and bulbar symptoms are now pronounced: The spastic-paralytic condition with muscular atrophy, increased reflexes, and reaction of degen-



eration in the extremities as well as in the trunk, prevents the voluntary change of position in bed. Mastication is feeble; there is facial paresis; diminished taste along the left half of the tongue towards the tip; marked dysarthria; vocal cord paresis; atrophy with paresis and contracture in the tongue. In addition to these exquisite symptoms of disease in the motor neurons there presents itself a nearly symmetric anæsthesia in the extremities (subjectively this was possibly noted already toward the end of 1889, but not demonstrated until in September, 1890), which is partial in the legs and feet as well as in the left ulnar district, but well nigh complete in the right ulnar area. This anæsthesia gradually fades away by the end of November, 1890; at this time partial loss of control of the sphincter ani is first noted; a little by little spontaneous, persistent pain develops first in the right, and, a few months later, in the left forearm along the ulnar nerves, and finally in the lower extremities also. In the May, 1891, there is found some loss of touch, pain, and temperature sensibility in the trunk and limbs; in November, 1891, the feces pass involuntarily; there is now concentric limitation of the visual fields and the eye-movements are jerky; there is further aggravation of the other symptoms detailed, both motor and sensory; there is slight delay of transmission of painful impulses. In February, 1892, cachexia has become marked; albuminuria and ordinary incontinence are now present, and temperature and pain sensibility is dulled in the face also; he cannot distinguish between sweet, sour, and salt; the sterno-cleido-mastoid and trapezii are also atrophic. In the trunk and limbs touch, pain, and temperature sensibility are further diminished. Contractures at the elbows and elsewhere are present. In March, shortly before death, there is double sensation and marked delay in transmission of impulses, both from face, trunk and limbs: a firm pinch being felt as a touch in 1-3 seconds and as pain in 6-8 seconds. Extreme anarthria, the mind remaining clear. Death, March 23, 1892, from exhaustion, four years and eight months after the appearance of the earliest distinct motor symptoms.

#### SUMMARY OF THE CHANGES.

The essential lesions may be summarized as follows:

Primary, chronic degeneration in the indirect motor neurons as shown by slight but distinct changes in the

cortex of the central convolutions and by degeneration in the pyramidal tracts from the internal capsule downward. Similar morbid changes in the direct motor neurons, namely, atrophy of marked extent in the bulbar nuclei and in the anterior horns of the spinal cord, degeneration in the bulbar nerves, in the anterior spinal nerve roots and in the mixed spinal nerves, in the cauda, the median and ulnar nerves, and also atrophy of the muscles of the tongue, larynx, and pharynx (other muscles not examined). Then there are quite identical lesions, though of less extent, in the direct sensory neurons: changes in the ganglion cells of the spinal ganglia, degeneration in the posterior spinal nerve roots and in Goll's columns: also, changes in the trigeminus sensory roots and the sensory part of the left trigeminus nerve (the Gasserian ganglia were not examined). In the indirect sensory neurons there are slight changes in the cells of Clarke's columns, but without degeneration in the direct cerebellar tracts.

There is also some thickening in the spinal pia and its vessels.

#### REMARKS.

The microscopic examination excludes all possible primary foci and diffuse lesions. Hence, the degenerations are not secondary. This observation is of interest in view of the attacks of unconsciousness mentioned in the clinical history as occurring for some years previous and up to the appearance of the early symptoms of the disease. These attacks are not explained by any of the microscopic findings.

Furthermore, the distribution of the lesions, as well as to a certain extent their histology, exclude the possibility of their dependence upon coarse arterio-sclerotic or syphilitic vascular changes; so that of whatsoever nature—certainly not syphilitic—the thickening in the vessels of the spinal pia may be, it cannot under any circumstances be regarded as playing any essential rôle in the production of the degenerations.

The degenerations are so systematic in extent and location that they correspond quite precisely with Flechsig's embryologic paths, as well as with the Wallerian tracts of secondary degeneration, both descending and ascending as far as the columns involved are concerned.

Consequently the degenerations in this case can be

defined as systematic and primary, depending in the main upon changes in the ganglion cells, in consequence of which the neurons became necrotic. In other words, the system lesions are the result of primary disease in both the motor and the direct sensory neurons.

The comparatively long duration of the sickness undoubtedly depended, to some extent, upon the absence of disturbances in the functions of the heart and of the lungs, which is rather remarkable in view of the degree of atrophy in the vagus nuclei. In connection with this the changes found in the left cervical sympathetic may be disposed of by saying that their relation to the other changes in the nervous system cannot be definitely specified, inasmuch as they form an exceptional condition without any corresponding known clinical manifestations.

As to the fundamental cause of the changes in the neurons, neither the clinical history nor the microscopic examination furnish any data. There are no indications of syphilis and no history of heredity. Malaria and small-pox are the only infectious diseases the patient ever had so far as known.

Should one believe with Strümpell, who has observed amyotrophic lateral sclerosis in two sisters,<sup>39</sup> that peculiar congenital and hereditary influences may play the same mystic rôle in individual instances of system diseases, even when no facts in the history point in that direction, as he regards them to do in his hereditary spastic spinal paralysis? The long duration of this man's disease would be one fact pointing in that direction.

Whatever the cause may have been, it had an apparently specific or selective effect upon the ganglion cells of both motor and sensory neurons. It was not coarse or intense enough to induce the ordinary phenomena of inflammation, and yet an extrinsic cause could reach the scattered cells only by way of the blood vessels. The only known form of chronic intoxication suggested by analogy is the syphilitic: the affinity of the syphilitic poison for the sensory neurons is generally accepted; its occasional appearance in an apparent etiologic relation to pure motor tract lesions not unknown, and, as already mentioned, there are quite a few cases of a somewhat similar combination of lesions as in this instance described in syphilitics (Leyden, Dinkler, Mayer, etc.). But further than the changes described in the sensory

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<sup>39</sup> *Neurol. Centralbl.*, 1893, No. 19.

tracts, the present case lacks such evident ear-marks of syphilis as lesions of the nuclei of the motor nerves of the eye, optic atrophy, etc. It is true that a few degenerated fibres were found in the third, fourth and sixth nerves, but evidently this degeneration did not depend upon marked nuclear lesions.

In so far as the case here recorded stands without any recognizable etiology, it corresponds with amyotrophic lateral sclerosis with bulbar paralysis, of whose etiology really nothing is known, and as a typical example of which the man's sickness seems to have started. After a period of about two years, during which the sickness had progressed from early indefinite symptoms to a quite typical clinical picture, sensory disturbances in the form of symmetric anæsthesia upon the extremities appear, to pass away again in a short time, when a general diminution in most forms of cutaneous sensibility develops and increases, together with lost sphincter control, but heightened reflexes, marked motor and bulbar symptoms, until death.

The motor and bulbar symptoms are quite readily accounted for by the extensive lesions described in the upper and lower motor segments, while the sensory disturbances find their explanation in the changes in the spinal ganglia, in the posterior nerve roots with probable degeneration in the sensory fibres, in the mixed spinal nerves, and in Goll's columns; and, as to the face, in the degeneration in the trigeminus roots and in the sensory part of the left fifth nerve; and these changes allow the inference that similar changes as in the spinal ganglia would have been found in the Gasserian had they been examined. The more or less isolated degenerated fibres from the spinal ganglia accumulate themselves in Goll's columns, in which the degeneration naturally is strongest in an ascending direction.

The absence of degeneration in other parts of the spinal cord, in which sensory paths go upward, point conclusively to the interpretation that the sensory symptoms were due to the changes in the direct sensory neurons, which possibly began in their more peripheral parts. The changes in some of the cells in Clarke's columns form an exception to part of the last statement, because these cells are regarded as the commencement of certain indirect sensory neurons, but there was no marked degeneration in the cerebellar tracts, although it is possible that a few scattered degenerated fibres

belonging to them might be running at the boundary between the cerebellar and the degenerated pyramidal columns.

Flechsigs<sup>39</sup> has called attention to the fact that in amyotrophic lateral sclerosis there is frequently some change in the cerebellar tracts as well as in Gower's columns, and Marie<sup>40</sup> also emphasizes the indistinctness of the outlines and extent of the changes in and about the pyramidal columns in this disease. Charcot and Marie,<sup>40</sup> Collins,<sup>41</sup> Dejerine and Hult,<sup>42</sup> v. Kahlden,<sup>43</sup> and others, all describe changes in Clarke's columns without corresponding degeneration in the cerebellar tracts and without sensory symptoms.

Any lengthy explanation of the peculiar and varying earlier manifestations of the sensory lesions—the temporary symmetric anæsthesia—the mechanism of the return of the sensibility, whether the underlying changes were first peripheral or first central, will not be attempted. Attention may be called, however, to this fact: that in accordance with the neuron doctrine parenchymatous changes due to various causes may locate themselves in any part of the neuron, most likely first where the influence of the ganglion cell is least felt. This must be quite true so long as the neuron is regarded as an anatomic and nutritive unit of which the ganglion cell is the essential centre. Hence, there may be partial neuron necrosis, and such partial neuron necrosis might very easily be mistaken for a genuine diffuse lesion—a neuritis, for instance, if the necrosis should be situated peripherally. The examples of "motor neuritis" in tabes dorsalis may be instances of partial motor neuron necrosis in connection with the changes in the sensory tracts. Joffroy and Archard<sup>44</sup> have a case of amyotrophic lateral sclerosis with a neuritis in the lower extremities, which they explain as due to a "spinal dystrophy." This case may very likely have been an instance of partial neuron necrosis. In the present instance a considerable number of the direct sensory neurons finally became totally necrotic.

This observation taken in conjunction with the instances of amyotrophic lateral sclerosis cited from

<sup>39</sup> Quoted by Goldscheider, *loc. cit.*

<sup>40</sup> *Loc. cit.*

<sup>41</sup> *St. Bartholomew Hosp. Rep.*, 1883.

<sup>42</sup> Virchow and Hirsch, *Jahresbericht*, 1886, Bd. ii.

<sup>43</sup> *Zeigler's Beiträge*, 1893, xiii.

<sup>44</sup> *Arch. de Med., exp. et d' anat. path.*, 1890, No. 3.

Charcot, Leyden, Maeli, Roveghi and Melotti as presenting system changes in the posterior columns, but without recognized sensory disturbances, would tend to show that this combination of changes in amyotrophic lateral sclerosis may not be so unusual as the name and the general idea of the disease may lead one to infer.

In addition to the unusual combination of system lesions presented by our case, the circumstances under which they are produced are also of interest: to the total necrosis in both motor neuron systems which appears as a typical amyotrophic lateral sclerosis—bulbar paralysis—a disease of definite entity, though unknown etiology, are added similar changes in the direct sensory neurons and, perhaps, in some of the indirect, so that finally there is presented a clinical picture in which both motor and sensory symptoms stand out clear and well defined, the motor predominating.

The unknown agent that first caused slow but progressive necrosis in the motor neurons was also capable of causing the same changes in the sensory in such a degree as to produce an exquisite example of combined system lesions.

The case consequently becomes a sort of connecting link between two groups of diseases in the nervous system that have usually been regarded as separate and distinct, namely, those in the motor segments, such as progressive spinal muscular atrophy and amyotrophic lateral sclerosis on one hand and tabes dorsalis, whose lesions involve the sensory neurons, on the other.

Finally, looked upon as a combined system disease, this case may be pointed out as illustrating a somewhat unusually complete combination of lesions. As shown in the introduction the vast majority of the combined diseases attack the posterior columns and pyramidal and cerebellar tracts—in a certain measure the indirect neurons—but in this case there is total neuron necrosis in both the motor and in the direct, as well as to a very slight degree the indirect sensory neurons.

EXPLANATION OF FIGURES.

FIG. 1. *Cell-forms from the layer of large pyramidal cells in the cortex of the central convolutions.*

1. Degenerated cell without nucleus and processes from the left arm centre.

2. Remnant of cell from left arm centre.

3. Atrophic cell without shrunken nucleus and corkscrew process from central part of the right ant. centr. convolution.

4, 5, 6. From lowest part of the centr. convolutions. 4. Granular mass. 5, Pyramidal cell in early atrophy. 6, Atrophic remnant.

Alcohol hardening, methylen blue staining. Zeiss' obj. 4 mm. apochrom. — ocul. 8.

FIG. 2. *From the hypoglossus nucleus* Pigmented atrophic cells, and slightly sclerotic stroma.

Weigert preparation. Zeiss' obj. 4 mm. apochrom. — ocul. 8.

FIG. 3. *Transverse section of the spinal cord at the fifth cervical segment.*

Degeneration in the lateral pyramidal, left anterior pyramidal and Goll's columns. Owing to incomplete decussation, the right pyramidal tract is a little smaller than the left. Weigert preparation,  $\times$  6 times.

FIG. 4. *Ganglion cells from the anterior horns of the spinal cord.*

1. From lumbar region: the nucleus absent, the body shrunken, the staining diffuse, some processes lost or changed.

2. Changed cell with demonstrable nucleus.

3 and 4. Pigmented granular forms from the cervical enlargement.

5. Dense and diffusely stained mass.

6, 7, 8. Smaller, round granular forms.

Alcohol hardened, stained in methylen blue, Zeiss' obj. 4 mm. apochrom. — ocular 8.

FIG. 5. *Transverse section of distal dorsal cord*, showing fusiform venous channel passing between the central canal and Clarke's column. Notice the very few ganglion cells in the anterior horns.

Alcohol hardened, stained in methylen blue,  $\times$  6.

FIG. 6. *First dorsal spinal ganglion.*

a, normal ganglion cell; b, shrunken cell.

Weigert preparation. Zeiss' obj. 4 mm. apochrom, — ocul. 8.

## ON A CASE OF ACUTE POISONING BY ARSENIC WITH SUBSEQUENT MULTIPLE NEURITIS.<sup>1</sup>

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J. W., 19 years of age, though a steel engraver by occupation, was in the habit of amusing himself by the practice of taxidermy. The shop in which he was employed was infested with rats, and he suggested to his employer that he had a remedy at home that would put an end to the rodent pest. He referred to a quantity of powdered white arsenic (more properly speaking arsenious acid) which he had recently purchased, and which he used as a preservative. He was also in the habit of taking "bromo-selzer" for headache. He filled an empty "bromo-selzer" bottle with arsenic for the purpose of taking it to the shop. On the way to his place of business he purchased a fresh bottle of the much advertised anti-cephalalgic. Upon reaching the factory he thoughtlessly gave his employer the recently purchased bottle, and retained that which contained the arsenic. In the course of the morning a slight headache caused him to think of his favorite remedy. He poured out a teaspoonful of arsenic (about seventy grams by weight), and attempted to dissolve it in a goblet of water. He observed that it did not dissolve or effervesce, and thought it rather remarkable. Nevertheless, he gulped down the contents of the goblet, and immediately perceived the grievous mistake he had made. He forced himself to vomit by thrusting the finger into the pharynx, but the vomiting was not very copious. He then went home and lay down. Three hours after the ingestion of the arsenic severe pains came on in the stomach; he began to vomit, and continued to do so for three days at intervals of five to ten minutes; nausea was present between the attacks of vomiting; food could not be taken, and he developed an intense, burning thirst. A physician was called who prescribed iron as an antidote, but this could not be retained in the stomach. The thirst was so intensely mad-

<sup>1</sup> Presented at a meeting of the New York Neurological Society, January 8, 1895.



dening that he attempted to leave the bed to relieve it, but he was so weak that he fell over in a faint. The pain in the stomach lasted for six days; it was somewhat relieved by morphine injections and the application of hot poultices to the epigastrium. The pulse was very weak. The urine was bloody and its passage caused considerable pain. The bowels at first loose, remained costive for four days. On the second day after the poisoning the patient's eyesight became affected. He could see only in the recumbent posture. Attempts to sit up would result in attacks of syncope. The disposition to syncopal attacks lasted about a day and a half, and was doubtless due to the weak heart action. Then deafness came on, and lasted about three days. There were continual tinnitus aurium, and hæmorrhages from the ears, followed by the discharge of pus. The ringing in the ears persisted for six weeks, then ceased, first in one ear and four days later in the other. On the fourth night after the poisoning the patient became delirious, and developed hallucinations of sight. This lasted but a couple of days. The tongue was blistered and swollen, the lips, eyes and rest of the face were œdematous. Excepting a few "white pimples" on the face, as the patient describes them, there was no skin eruption. He had severe burning headaches, which were not relieved by ice-applications. On the seventh night he entered into a condition of collapse, but rallied the next morning. On the ninth day the pain in the stomach ceased, and the patient could tolerate a little food. On the eleventh day he was able to leave his bed and walk about. He had lost twenty pounds during his confinement. Nine days after leaving the bed (end of third week after the poisoning) severe, continuous, burning pains manifested themselves in the legs, preventing sleep. Three days later the pains appeared in the feet, which became swollen. The pains remained in the feet for nine weeks. He had never had pain in the arms, but some tingling in the hands. Two months after the accident he entered the New York Hospital for treatment, where he remained for eleven days. He says he was able to walk to the hospital, but when he left he had to be taken home in a coach. This inability to walk caused him to resort to crutches, which he was using when he came to Dr. Hammond's clinic on October 15, 1894. He had never seen double, the facial muscles had never been affected, nor had there ever been any speech disturbance. An examination of the patient showed the following

condition: His gait is that of an ataxic; the legs are lifted high, to enable the toes to clear the ground, and are brought down with a stamp. He walks decidedly better with the shoes on than off. The feet droop; they are cyanosed and cold; there is marked tenderness to pressure of the feet, legs and thighs; involuntary contractions and twitchings are seen in the calf muscles. The muscles of the leg are atrophied. The calves measure ten and one-fourth inches in circumference, the thighs at their lower third measure twelve inches, and at their upper third fifteen inches. The thigh muscles have preserved their coarse strength. The weakness affects the parts below the kneejoint. With reference to the reflexes the examination shows absence of the foot, the patellar, and the cremasteric reflexes. The abdominal, epigastric and gluteal reflexes are present. Control over the contents of bladder and bowel is normal. As regards sensation, tactile anæsthesia of the feet is found extending to a point immediately above the ankle joint; two inches above this is an area of hypæsthesia. Both lower extremities show pretty generally hyperæsthesia to pain. On testing with test tubes containing hot and cold water, the feet are found to be markedly anæsthetic, whilst the lower half of the legs are hypæsthetic. There is also marked anæsthesia of the legs to faradism and galvanism. The patient is able to slightly extend the feet, but finds it impossible to flex them. The electrical tests show R. D. for both the faradic and the galvanic currents. In the upper extremities it is the hands that are mainly affected. Owing to the marked atrophy of the interossei, the lumbricales, the muscles of the thenar and hypothenar eminences, the movements of the fingers are greatly impaired. Flexion and extension of the hands and forearms are good. The atrophied muscles show the reaction of degeneration. Sensation to touch in the hand is considerably diminished, some areas being anæsthetic. The reflexes in upper extremities are absent.

The muscles of the trunk show no effects of the poison, nor are there any disturbances referable to the central nervous system.

The treatment consists in the application of electrical currents (the static and the induced), and the administration of strychnine sulphate, of which he is at present taking one-twelfth grain, three times daily. Under this treatment he has improved, being now better able to use the hands and feet than formerly.

## Asylum Notes.

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### POINTS OF VIEW IN CLINICAL STUDY OF PSYCHIATRICAL WORK.

It is manifest even on superficial study that physicians studying mental troubles may be divided into two classes: the asylum medical officers and the neurologists. The able paper of S. Weir Mitchell has called prominently to notice that there is this difference. This paper has called forth, besides the letters by which it was accompanied, many comments from the medical press, which have been usually of a commendatory character.

Much as we are interested in these comments, it is only of the "principle of judging" of which we now comment. The point of view, in looking upon mental troubles, is, if we mistake not, rarely fully apprehended. Neurologists do not see insanity in the same way, do not see the same cases, do not have the same prognoses as asylum officials. Medical officers in asylums, on the other hand, do not, many of them, get the same detailed histories and the personal interest in particular cases that the neurologist does, and is apt to tend toward routine and to lose interest too early—is apt to custodially serve, and to lose the patient's individuality in the mass. The best hospitals for the insane overcome this tendency fairly well, but the tendency is always present, and is fostered by the seclusion, lack of competition, and a too keen skepticism concerning therapeutic efforts.

Of the two points of view, the asylum officer has the most comprehensive. But the neurologist, by his keener medical study, unencumbered by custodial and business cares and habits, will often be ahead. Let us look at some of these points of difference. A prominent neurologist of this country is said to have pointed recently to the proportion of melancholias of a certain asylum,

marked "cured," and to have asked why his own list showed a much larger proportion of recoveries.

He undoubtedly was correct as regards having the most recoveries. But it is also undoubted that he ought to have had this greater proportion. He saw cases early, milder cases, and cases which would be pecuniarily able to receive the benefit of early and energetic effort and painstaking care. The asylums received the remainder—those of longer duration, of extreme melancholia, of neglected and poverty-stricken families, of gross syphilitic and alcoholic recklessness, and particularly of defective and degenerative tendencies.

It is, though anomalous in sound, a true statement, that in many ways the best place to study insanity is outside the asylum walls. It is true that one needs a thorough living knowledge of the gross insanity as a basis for such study, but, given that, he can get his most valuable hints from those nominally sane, and in the beginnings of insanity. In the density of the mental cloud of the chronic cases of insanity as the disease progresses all outlying lines are lost—indeed, are to a large extent obliterated. But, in the early stages, the case is alive and bristling with prominent points and suggestions.

For example, headaches, their character, their times, causes and localities, can be studied early in the history of the case. Later they are obliterated; rarely are they to be found as symptoms even of curable insanity in an asylum. All bodily complications are subject to the same obliterating change.

When we say that the most interesting data concerning insanity are found in the so-called sane, we mean in detail simply this: First, the most interesting data, symptoms and complaints of mentally diseased patients come usually before they are so dulled or deranged as to receive asylum care. Second, in the moods of the sane, in the hysterias, in the vacillations of the menstrual periods, in the perversions of pregnancy, in the transient hallucination of drugs or of other slight disorders, in the phenomena of drunkenness, in the symmetrical decline from chronic drinking, in the many cases of slight feebleness of mind which still remain this side of imbecility, in the delirium, in imperative ideas, in the morbid, half-outlined ideas of persecutory nature, in the hypochondriacs, neurasthenics, or slight melancholiacs, in the eccentricities, in the mental conditions of those not com-

pletely recovered from insanity, and in many other states that could be enumerated, we can most accurately and most easily read the mental changes. Of these cases, perhaps nearly nine-tenths are to be found outside of asylum practice.

Not that, in accord with the recent sarcastic remarks of W. P. Howie (*Journ. Amer. Med. Assn.*, Dec. 15, 1894), we would call all genius, all immorality, all drunkenness and all extremes as "insanity." Indeed, we would carefully avoid this. Yet these mental states enumerated are the most easily read and studied nevertheless, and the line of march of modern thought in the study of these lines will rarely go to excess, for the reason that the powerful instinctive self-defense of society, especially in legal and criminal cases, is steadily repelling such advances.

Hospital care of the insane we do not here comment upon, but would like to appeal to the conviction of each of our readers as to the existence of the differences of the field for study and opinion as here outlined,

### COMMENTS ON HOSPITAL REPORTS.

Each of the approximately 120 public hospitals for the insane, of this country and Canada, issues every one to two years a report of its year's progress. Such reports are addressed usually to the governor and legislative body of the State. Inasmuch, however, as it is the only report issued, and as probably its chief circulation is an exchange with other hospitals and among physicians, and as, moreover, this is partially recognized by the publication of tables of clinical data, together with occasional clinical and pathological notes, we have held that it is fitting to make them a medium for consideration of clinical study.

If not in this report, then surely in a special one, each hospital would do well to issue a report of medical, pathological and psychological work and study. This would at once stimulate competition, and divert from the strong trend of business, political and custodial administrative work.

**Each report notes** its wants for the next year and its buildings for the past year. Each one has some plans for buildings or construction. Each one mentions amusements, occupations, etc., in methodical manner. Of

these ordinary custodial ideas, local in character, no comments need be made. Of several ideas more modern and administrative we note that "congregate" dining-rooms and cottage systems of building seem to have lost much prominence. On the other hand, baths and hydrotherapeutic measures are receiving more prominent consideration.

The Milwaukee County Asylum (Dr. White, Superintendent); the Binghamton State Hospital, New York (C. T. Wagner, Superintendent); the Warren State Hospital, Pa. (M. G. Gutk, Superintendent); Jacksonville Asylum for the Insane, Illinois, have all reported Turkish baths or some equivalent. The two Minnesota hospitals are also having these baths put in. The Warren Asylum, in its last report, declares it to be useful, and asks for an extension of the system to the female part of the hospital. "Hygeia Hall" is with them a special building for the reception of new cases, which will contain the special bathing facilities for the female population.

**Dr. Walter Channing** (*Journ. of Insanity*, October, 1894) makes reply to Dr. S. Wier Mitchell's criticisms of last summer. He does it, probably to the surprise of many, by trying to uphold the superintendent's position as a custodial worker as the correct one. He says "they are not neurologists or psychologists, and should not set out to be." He assumes that their time must necessarily be taken up with custodial work, and that if a neurologist were put in charge he would have to devote himself in like manner or inevitably fail.

*We do* not think these statements in good accord with superintendent's views as outlined elsewhere. But even if they are acknowledged as correct, we would suggest that it is not the superintendent's care over diet, nursing and ward furnishing that is under criticism, so much as the far greater outside interests. For example, the running of an immense farm is quite foreign to the patient's care, even though it furnishes them with supplies. Still farther, admitted that he must have all these immense interests, is that any reason why another man should not then have charge of those clinical, pathological and psychological investigations, which all admit cannot be done in addition to the business. Surely such a department exists and ought to be cultivated.

**Dr. Irwin H. Neff** (*Journ. Insanity*, January, 1895) writes an interesting history of thirteen cases of hereditary ataxia, all of one family, distributed through four generations. It came on from the ages of fifty-six to seventy-five, and seemingly always in about the same manner. Four of the cases had insanity of the form dementia. The invasion was slow. There were articulatory troubles current. As with others likely who have studied hereditary chorea, the many parallel lines in which the two troubles travel strike us at once. The form of motor disturbance seems the chief difference between the two.

**The death** of Dr. J. B. Andrews, upon August 3 last, deprived the Medico-Psychological Association of one of its most prominent members in this country. He has been superintendent of the Buffalo State Hospital since 1880, and has been in insane hospital work since 1867. He was president of the section of Psychological Medicine and Nervous Disease of the Ninth International Congress of 1887. He was president of the Medico-Psychological Association, and represented it during the World's Fair year.

**Dr. George C. Palmer**, who died August 17, 1894, was another of the older members of this association. In 1864 he received an appointment as assistant physician in the Michigan Asylum for the Insane at Kalamazoo. In 1872 he was made assistant superintendent; in 1878 he became superintendent, and in 1891 he resigned to take charge of a private institution. He always commanded respect and affection, and died honored and esteemed by all with whom he had dealings. Both he and Dr. Andrews are examples of that desirable selection of a steady, persistent life-calling, which is in contrast to some appointments to a superintendency of men who never have had hospital experience.

**The Morningside Mirror** is the name of a small paper published monthly at the Royal Edinburgh Asylum; whether by patients, employees or physicians is not stated. It has no column for items or news, but is filled with brief essays on such subjects as "Golf," "Curling," "The Opening of the New Craig House," etc. This latter was evidently made quite an important event, being celebrated by formal exercises, including explanatory speeches and a description of its aim by

Dr. Clouston, and ending with a ball, in which patients and employees took part. The Duke and Duchess Buccleuch were selected for the ceremony of formally declaring the house opened. We note that Dr. Clouston enforced again the idea that the whole institution was a hospital. We also noted in the November number an account of the departure of Drs. Elkins and Wilson, two of Dr. Clouston's assistant physicians who seemingly leave at the same date. [The *Morningside Mirror* is written, edited, printed and published by the patients of the Morningside Asylum.—Ed.]

**The Illinois** State Medical Society proposes a law to be urged upon the Legislature providing for the appointment by the judges of the Circuit and Superior Courts, of persons who will act as expert witnesses in medical and other sciences in giving opinions upon the evidence as presented in hypothetical form in criminal and other cases pending before such courts. The term of appointment is to be for one year. The judge may summon as many as three witnesses. They shall be subject to cross-examination, but only on subjects embraced in their opinion.

**The Essex** County (Newark, N. J.) report mentions its day school for patients, established in 1893, as quite successful, and is still exciting great interest. The Middletown (New York) Asylum for the Insane noted in 1888 the establishment of a school so extensive as to take up besides the common branches, book-keeping, biography, composition, drawing, grammar and history. Many hospitals have had various "meetings for instruction," but so extensive a regular school is rare. Dr. Lalor, of Richmond Asylum, Dublin, first made prominent this element, claiming its indirect effect as an occupation and mental stimulant to be good.

**On the** morning of January 4, a fire occurred in the large hospital for insane at Anna, Ill. The destruction of the older parts of the building is reported as quite complete. No lives were lost and no patient escaped, which is quite wonderful, considering the confusion and haste. This will relieve the event of most of its horror in the public mind. Fire was first found near the roof, but the origin is reported unknown.

**The establishment** of a second association of superintendents, called "the Association for Southern Hospitals for the Insane," has been announced. We are



not informed as to its especial aim. Its first meeting was announced for November 20, 1894, but owing to various causes and the political pre-occupation of the month, the meeting was postponed, subject to the call of the committee.

*In Kansas* a laboring man, named McDonald, killed another man named Pelton. In defence it was claimed that he was under the hypnotic power of a third man, an enemy of Pelton's, named Gray. The jury found Gray guilty and acquitted McDonald. This seems very dangerous doctrine, and though extreme, is but one sign of the wave of speculative popular interest in hypnotic influence that is now abroad in some of the western states. The case, we understand, has been appealed.

*The Engineering Record* of December, 1894, contains a very full description of the rain bath system at the Utica Asylum, New York. Inasmuch as they are shown to have a special building, and thirty-nine different sprays which can go at once, it is probably as extensive and complete a system as at any hospital in the country. The writer's estimate of the running capacity of the system, assuming, as it does, that the whole thirty-nine sprays are running at once, would seemingly assume that the patients are all sane enough to bathe themselves, or that a very large force of employees would be needed. Probably not one-half of the patients at any hospital can be trusted to bathe themselves.

*The January* number of the *Journal of Insanity* just out is both very interesting and very creditable to its new management. The Journal for some years has been steadily improving in our opinion, and the advances promise to be fully as rapid in the future. The present number, moreover, is nearly all on clinical and pathological lines. The "Abstracts and Extracts" of current literature are becoming very thorough and comprehensive.

*The Kankakee Hospital for the Insane* (Ills.), held last summer a Summer School for special instruction to medical men, in mental diseases and pathology. Dr. Clarke Gapen is the superintendent, and the course was seemingly conducted by Dr. Adolph Meyer, Pathologist to the Hospital.

*The laboratory* of the McLean Hospital for the Insane (Mass.), is fully outlined in the January number of

*Journal of Insanity.* There is an examination room, a chemical room, an apparatus room, a microscopical and photograph room, a mortuary room, an autopsy room. Dr. Hoch, having spent two years in Germany in special study, makes a specialty of the research work here. The assistant physicians and superintendent each work along special selected lines of investigation. This outlines a very desirable trend for hospital work.

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**"Emaciation Insanity."**—Brissaud and Soques. (*Delire de Maigreur. Nuov. Iconogr. de la Salpetriere*, December, 1894). With this title the authors propose to designate what has generally been termed hysterical anorexia. They contend that in the light of recent investigations, which have shown hysteria to be a mental disease, this current term is inaccurate and not sufficiently expressive. A little objection may be made to the substitutes, hysterical inanition and sitieirgia, proposed by Lasègue and Sollier respectively.

In all cases alike, whether the appetite is lost or not, the emaciation is the sequence of a conscious or subconscious imperative conception (*idée fixe*). They support this thesis by a very full presentation of the case of a young girl of nineteen, distinctly hysterical, in which the development of the idea to grow thinner is carefully traced from its inception, a simple desire for a better form and to escape the raillery of her companions, up to the development into a complete "possession." For three years, with only short intermissions, she had been unable to retain food, and had wasted to a mere skeleton, her weight having fallen from sixty to twenty-nine kilogrammes. The skin was brown, wrinkled and flaccid; the hair dry, with spots of alopecia; the nails furrowed and irregular; the temperature subnormal. Isolation, mental treatment and feeding added thirty-one kilogrammes to her weight in three months, at the end of which time she was entirely normal in every way. In the treatment the authors lay stress upon the necessity of substituting for the imperative conception of emaciation one of increase in weight. They further call attention to the fact that in animals, if emaciation exceeds six-tenths of the initial weight, restoration is impossible, and infer that a similar law holds good for man, so that there is nothing to prevent a fatal termination in extreme cases of this hysterical affection, and several instances of such termination are cited.

PATRICK (Chicago).

# Periscope.

## PHYSIOLOGICAL.

**Some Disputed Points Regarding the Doctrine of Central Localizations.**—By J. M. Charcot and A. Pitres (*Archives Cliniques de Bordeaux*, No. 9, September, 1894).

### I.

#### DOUBTFUL OR INSUFFICIENTLY DEMONSTRATED MOTOR LOCALIZATIONS.

The only positive motor centres known to-day are those for the extremities, face and tongue.

Some authors believe as definitely localized other motor centres which we believe insufficiently demonstrated: 1. Phonation. 2. Rotation of the head. 3. Conjugate deviation of the eyes. 4. Elevation of the superior eyelid.

##### (a) *Cortical motor centre of the larynx.*

Ferrier '73, Duret '77, Krause '84, by experiments upon dogs, found that irritation and extirpation of the antero-inferior part of the pre-frontal gyrus affected, respectively, the vocal function of the animal. Unilateral extirpation of this part caused but little laryngeal trouble, but bilateral extirpation rendered the animal incapable of barking. Masini, '87, Semon and Horsley, '89, confirmed these observations upon monkeys. Observations are too rare to establish anything with certainty in man.

The authors are able to find only four cases reported with persistent aphonia where autopsies have been made, showing circumscribed cortical lesions. In two of these cases the lesions involved the right lobe alone.

Seguin advanced the hypothesis that the cortical laryngeal centre is in the foot of the right third frontal convolution. So many cases of destruction of this part without aphonia have been observed that the hypothesis is not accepted.

The opinion that the centres are bilateral and symmetrical, as in monkeys and dogs, located at the inferior extremity of the region of Rolando in the immediate neighborhood of the motor centre of tongue and inferior facial, is more reasonable but not demonstrated.

##### (b) *Motor centre of the head.*

Ferrier, in electrifying posterior part of the first frontal convolution of monkeys, obtained rotation of head. Beevor and Horsley confirmed this. In man, destructive lesions of foot of first frontal convolution give no paralysis of muscles of neck, while several cases are reported in which foot of first frontal was altered or destroyed unaccompanied with paralysis of muscles of neck.

##### (c) *Centre for conjugate deviation of the head and eyes.*

This symptom complex has been carefully studied, especially by Landouzy and Grasset. These authors localize a centre for these movements at the level or in the neighborhood of the infra-parietal lobule. Our observations do not lead us to accept this centre as demonstrated.

Wernicke and Henschen believe the centre to be located in the infra-parietal lobule. Wernicke analyzed published reported cases and concludes :

1. Conjugate deviation of the eyes is always in relation with a lesion of the infra-parietal lobule or of fibres emanating from it.

2. Lesions of the infra-parietal lobule always produce conjugate deviation of the eyes, if only transitory.

Charcot and Pitres believe the following invalidates Wernicke's conclusions :

1. We have reported cases where lesions of the infra-parietal lobule were not accompanied with conjugate deviation of the eyes.

2. Conjugate deviation of head and eyes may exist without lesion of infra-parietal lobe. A case is cited—Right hemiplegia, conjugate deviated eyes toward left. Circumscribed softening of left ascending convolution alone.

(d) *Centre for elevating the superior lid.*

Exceptionally in hemiplegia is found a paralysis of the superior eyelid. Grasset and Laudouzy place a cortical centre for same in the angular gyrus, and offer a number of corroboratory observations :

1. Softening of the left angular gyrus—incomplete right ptosis.

2. Softening of left angular gyrus—persistent ptosis right superior lid.

3. Softening of left infra-parietal lobule—right ptosis.

Charcot and Pitres hesitate to admit a definitely localized centre on following grounds :

1. Large number of observations where no ptosis existed, yet autopsies showed extensive alterations of the angular gyrus.

2. Ptosis is described in a large number of cases where the lesions reach beyond, and often far beyond, the region of the angular gyrus.

We saw it exist in two cases, with cortical softening of the inferior extremity of the ascending convolutions. In a third case, with softening of ascending frontal and base of second frontal.

## II.

### DISTURBANCES OF SENSIBILITY IN THEIR RELATION WITH LESIONS OF THE CORTICAL MOTOR ZONE.

Hitzig offered the hypothesis that troubles of motility caused by destruction of the cortex may be due to perturbations of muscular consciousness.

This hypothesis was accepted with modifications by a number of physiologists—Notnagel, Schiff, Munk, etc.

Notnagel explained motor paralysis of cortical origin by perturbations of muscular sense.

Schiff believed lesions of zone called motor caused tactile anæsthesias, and these, secondarily, ataxias of motion rather than real motor paralysis.

Munk generalizes. He thinks that the brain has no direct action upon motility; the motor region is the region where end and are elaborated the sensations of contact, pressure, temperature, position, etc., of various parts of the body. If lesions here cause perturbations of motion, it is simply because they cause total or partial loss of kinesthetic sensations, which excite and direct motility.

Bastian agrees, denying existence of any motor centres. He thinks the Rolandic area is formed by an aggregation of sensory centres, the activity of which excite voluntary motor centres in pons, medulla and spinal cord.

Ferrier, Bechterew, Horsley maintain, on the other hand, the doctrine of the anatomical and functional separation of motor and sensory centres. They affirm that cerebral mutilations limited to the Rolandic area do not cause *per se* any sensory perturbation.

Clinicians do not agree any more than physiologists. Tripiér, Ballet, Exner, Starr and others who have noted frequent co-existence of sensory perturbation with motor paralysis of cortical origin, are inclined to admit hypothesis of superposition of motor and sensory areas.

Inversely, other observers, having found sensibility intact in a large number of cases of hemiplegia dependent on lesions limited to Rolandic convolutions, do not accept theoretical deductions. What are the facts?

Ferrier cites 284 cases: Sensibility not mentioned, 100; sensibility intact, 121; sensibility altered, 63. Motor paralysis of cortical origin exists two out of three times with no sensory perturbation.

In twelve observations brachio-facial monoplegia we have found: Sensibility intact, four; sensibility altered, three; sensibility not mentioned, five.

In twenty-two cases brachio-crural monoplegia: Sensibility not mentioned, thirteen; sensibility intact, seven; sensibility altered, two.

In twelve observations, crural monoplegia: Sensibility not mentioned, five; sensibility intact, five; sensibility slightly altered, one; diminished tactile sensibility, entire body with preserved pain and temperature sense, one.

Finally, eight observations, facial monoplegia: Sensibility not mentioned, four; sensibility intact, two; sensibility altered, two.

In one of the latter two cases anesthesia extends beyond face to upper part right side of trunk; in the other case it was general.

Sensory phenomena not only are not superimposed upon motor, but they are transient. All who have studied them agree upon this. Shall we say they are capable of displacing themselves from one moment to another, of disappearing and reappearing from slight causes if they depend on the destruction of corresponding perceptive centres?

It remains to explain why alteration of motor zone in about one-third cases are accompanied with disturbances, cutaneous or muscular sensibility. Legroux and de Brun refer them to circulatory disturbances which are but beyond points in which paralytic lesion is located.

In our opinion the anesthetics which sometimes accompanies the motor paralysis of cortical origin are mostly functional, analogous, if not identical, with hysterical anesthetics—are superadded phenomena, accidental, not depending upon the lesion of the Rolandic region and playing no pathological part in the production of the paralytic symptoms.

### III.

#### LIMITED ATROPHIES OF THE MOTOR ZONE CONSECUTIVE TO OLD AMPUTATIONS OF THE EXTREMITIES.

We know that long after the amputation of an extremity there results a more or less complete atrophy of the corresponding spinal cord segment.

Does atrophy occur in the brain tissue?

Dickson found no atrophy of the brain in four subjects where amputation had been performed, from two to fifty-three years before death.

Vulpian was not more successful.

Later observers reported changes, but many observations are valueless since the amputation followed congenital deformities and acquired atrophies of the limbs. To be of any value we must limit our study to cases in which cerebral atrophy, if it exists, can be traced to loss of limb.

Thirty-seven cases of old amputations are cited: Atrophy more or less marked in motor zone of opposite side in twenty-two cases; **unchanged** in fifteen cases.

Analyses of these cases does not make it clear why atrophy in **some** and none in others. The analysis seems to demonstrate that the

cerebral atrophy is not the result of an ascending degeneration. The amputation of a limb is always followed by a unilateral atrophy of the cord segment corresponding to limb amputated. Above this region the gray substance and the white columns do not show alteration.

These atrophies are not constant enough nor sufficiently circumscribed to aid in the determination of functional topography of the cortical motor zone

ELLIOTT.

**On the Rule of the Facial Nerve in Secretion of Tears.**—By E. Jendrassik. (*Revue Neurologique*, No. 7, 1894.) In two cases of facial paralysis Goldzieher observed that the eye of the affected side remained dry during weeping; he therefore concluded that the lachrymal glands were supplied, not by the trifacial nerve, as is usually taught, but by the facial. Jendrassik, from observations on four cases of facial paralysis, inclines to Goldzieher's opinion. According to J., the lachrymal gland is supplied by the facial nerve as follows: Fibres are given off from the trunk of the facial nerve, as high up as the geniculate ganglion; these fibres run along the great superficial petrosal nerve to the spheno-palatine ganglion; from here one group of fibres is distributed to the soft palate, whilst another unites with the second branch of the trigeminus, another runs into the orbital nerve. The orbital nerve unites with the lachrymal nerve, and the combined nerves give off filaments to the lachrymal gland.

MEIROWITZ.

#### ANATOMICAL.

**The Employment of the Weigert-Pal Staining Method for the Central Nervous System Which Has Been Hardened in Formalin.**—Magnus (*Neurolog. Centralb.*, Jan. 1, 1895).

Formalin has shown itself to be one of the best hardening agents for macroscopical purposes and nuclear stains. The drawback to it for general use has been, heretofore, that nervous tissue so hardened would not accept the Weigert stain. Dr. Magnus has overcome this difficulty, and has succeeded in staining specimens excellently by employing the following method: After the spinal cord has been in a one-half per cent. solution of formalin for from two to four weeks, small pieces one-half cm. thick are cut off and placed in a solution of Müller's fluid in a bread oven kept at 37 C. for a week. After this they are a day in 95 per cent. alcohol, and then for a day in absolute alcohol, and then follows the imbedding in celluloidin. After the piece has been cut on the microtome the sections are placed again in Müller's fluid, and then put in the bread oven for from a day to a week. The sections are then quickly washed in alcohol and put in the Weigert-Pal hematoxylin solution, where they soak for at least two days. The stain which he has thus obtained is very distinct. The myelin sheaths are beautifully blue, and all the degenerated tissue is decolorized. The ganglion cells are very distinct, and their nuclei show clearly.

J. C.

#### PATHOLOGICAL.

**A Case of Extensive Ependymal Glioma of the Cerebral Ventricles.**—Pfeiffer. (*Deutsche Zeitschr. f. Nervenheilk.*, fifth volume, sixth part.) A thirty-two year-old cigar maker, who had always been healthy, began a year before to have twitchings of the head and eyes, these gradually increased in intensity, and were followed, after four months, by marked protrusion of the eyeballs. These were accompanied by psychical depression, slight degree of stupor, headache without definite localization and clonic convulsions in a part of the neck and face muscles. Later, right side Graefe's symptom, nystagmus, pupils equal, reactions normal, diminution of acuity of vision, ophthalmoscopically, retinitis albuminurica. Tremor of the hand, but not inten-

tional in character. Slowing of the pulse and attacks of vomiting when the headache was very severe. Right patellar reflex lost, left weak. Great variation in the symptoms from day to day. In the further course of the disease there appeared progressive diminution of sight, retinitic atrophy of the disc, double abducens paralysis, dilatation of the right pupil with slow reaction. Convulsive drawing of the face toward the right side, high degree of stiffness on attempts at movement, and pain of the cervical portion of the spinal column. Increasing stupor, slight attacks of unconsciousness, great slowing of the pulse, Cheyne Stokes respiration. Double-sided ptosis, great limitation of the movability of the eyes, deviation of the tongue to the right. Incontinentia urinae et alvæ.

Autopsy showed the ependyma of both lateral ventricles had lost its glistening appearance, and presented a grayish white and red swellings. These swellings were in the form of compact hard granulations in some places, while in other localities there was a more diffuse infiltration and a lessened consistency. Similar conditions were found in the extensions of the third and fourth ventricles, and in the aqueduct of Sylvius. Histological examination of the tumor masses found in the ventricles left the matter in doubt as to the real nature of the new growth. In some sections it resembled a glioma, in others a mixed form, glio-sarcoma. Considering the extensive and diffuse infiltration, the gradual development of the disease, the fine fibred structure of the supporting tissue, its peculiar light colored appearance, its vascularity and the retrogressive metamorphosis that was present, the author was struck with its similarity to the process that goes on in gliosis spinalis. J. C.

**A Case of Glioma in the Dorsal Portion of the Pons.**—Jolly. (*Archiv. f. Psych. und Nervenheilk.*, vol. xxvi., part 3.) The history of the case reported is briefly as follows: Duration of disease, less than two years. First symptom, left side facial paralysis. A month later vertigo, vomiting, and trouble in swallowing which disappeared after a week and did not return for three months. At this time subjective disturbance of vision, which perhaps depended on a paralysis of the muscles for winking of the left side. Simultaneously with this conjugate eye muscle paralysis developed; weakness of the muscles of mastication on the left side, and anesthesia in area supplied by the left trigeminus. A little later, weakness and lessened sensibility of right arm, which condition remained to the end. Later, similar disturbances in the left leg, then continuous numbness of sensibility in the right leg. The paralysis of the muscles of winking of left side was permanent till death, and toward the end a weakness of the same muscles on the right side was seen. Frequent attacks of weakness and vomiting, but no convulsions. Absence of choked discs. Diagnosis was new growth in the region of the pons, and as left facial paralysis was associated with conjugate deviation of the eyes to the right, it was considered that the tumor in its development had involved the nuclei of the facial and abducens. At the post-mortem, the cerebellum was pushed up, and under it, springing up from the bottom of the fourth ventricle, was a very large, grayish-white tumor; toward the cerebrum it was traceable to the aqueduct of Sylvius and spinalwards to the calamus scriptorius. The tumor lay substantially in left side of the bottom of the ventricle; apparently did not break through the ependyma of the ventricle, but to arch it up the raphe was pushed over to one side. The spinal end of the tumor, like a small plug, extended down and against the medial side of the left corpus restiforme. The trigeminus, as well as the facial and abducens, were somewhat flattened and transparent. The other cranial nerves, including the oculomotorius and trochlearis, seemed normal macroscopically. Microscopically it was found that the tumor had its greatest extent in the region of the origin of the facial and abducens nerves, and that it had destroyed the nuclei of those nerves not only on the side where it lay,

but it penetrated to the right through the raphe and infiltrated the abducens nucleus on that side. In its cerebral and in its spinal extension it limited itself rather completely to the left side, and involved on this side the trigeminus, the motor nucleus and the greater part of the sensory nucleus of which was destroyed. The greater part of the glossopharyngeal and hypoglossus nuclei were infiltrated and nearly destroyed by the tumour. The hypoglossus nuclei, in its proximal portion, showed in parts very marked changes, and as it was followed into the cord it became more and more normal. The tumor was a pure glioma. J. C.

**The Primitive Lesion of Tabes.**—Nageotte. (*Société de Biologie*, November 10, 1894.) The writer believes that there exists in tabes an intense perineuritis, which begins at that portion of the posterior roots which is situated between the ganglion and the point of entrance of the root into the sub-arachnoidian space. In the beginning this inflammation partakes of the nature of an embryonic process, while in the more advanced stages it passes in to a fibrous state. This inflammation is primitive, and leads up secondarily to degeneration of the posterior root fibres. The inflammation invades equally the anterior roots at a similar level, but the nervous tubes of these roots resist infinitely more the interstitial process than the posterior roots. Nevertheless it would be possible to invoke this lesion as a cause of the peripheral motor neuritis, at least in certain cases. The roots in the nerves in paralytic tabies do not differ from those in pure tabes in this regard. This conjunctive lesion is of the same order as those of the brain in progressive general paralysis. There exists no histological argument which prevents the admission that this embryonal lesion, afterwards sclerotic, may be syphilitic. J. C.

#### CLINICAL.

**The Reflexes in Total Transverse Lesions of the Spinal Cord.**—Gerhardt. (*Deutsche Zeitschrift für Nervenheilkunde*, Bd. 6, p. 127.) Bastian's assertion that a total transverse lesion high up in the cord abolishes the knee-jerk is well known, and has received the unqualified support of Bowlby, Thorburn, Bruns and others, although all are not satisfied with his explanation, which really comes from Hughlings Jackson, that it is the cutting off of cerebellar influence that makes the knee-jerk impossible. Gerhardt's case, which he considers as positive evidence against the view of Bastian, is a boy of seventeen, whose trouble began with a feeling of weakness in the right foot, gradually became paraparetic, in sixteen days was unable to walk, and a month later had become completely paraplegic. Sensation gradually diminished to a level above the umbilicus, but was not completely abolished until two years later. The knee-jerks were greatly exaggerated up to the fourth year of illness, when they began to diminish; by the end of the year were completely absent, and were not to be obtained up to the time of death, about five years from the onset. The superficial reflexes were retained, but they gradually altered in character, becoming very slow. The autopsy revealed an angioma extending from the fifth to the eighth dorsal vertebra, which had compressed and completely destroyed the cord in this region. He explains the loss of knee jerk by referring it to the marked contractures which had developed, with supposed changes in the muscles and joints. [Such changes might possibly be an adequate cause, but it seems fully as reasonable, if not more so, to assume that this slowly-growing tumor, slowly growing as shown by the long retention of sensibility, only accomplished the complete destruction of conducting paths in the cord at the end of three or three and a half years, and that from this time the tendon reflexes were lost.]

NOTE.—Since the above was written the review of Bruns has appeared (*Neurol. Centralb.*, January 15, 1895), in which he essentially agrees with the foregoing. PATRICK (Chicago).



**Apoplexy in its Relation to the Temperature of the Body, with a Consideration of the Question of Heat Centres.**—C. L. Dana. (*American Journ. Med. Sci.*, June, 1894.) The conclusions reached by Dana concerning apoplexy and thermogenesis are: 1. That all intracranial hæmorrhages, whatever their lesion, are much more apt to be accompanied with immediate disturbances of temperature than are necrotic processes from embolism and thrombosis. These temperature disturbances in hæmorrhages are, in rare cases, a sudden initial fall; then in almost all cases, except where the lesion is small, there is within a day or two a rise of temperature of from one to three degrees. On the other hand, in acute softening this initial fall and early rise do not occur unless the process is very extensive or involves the pons. 2. In apoplexy due to hæmorrhage, the temperature is greater upon the paralyzed side than on the normal, the difference averaging about one degree. In acute softening this unilateral difference of temperature does not occur or is extremely slight. 3. The rise of temperature due to apoplectic lesions depends more upon the extent and nature of the lesion than upon its location. Lesions of a hæmorrhagic character in the cortex, however, are especially apt to cause a rise of temperature. Lesions in the pons also, either of hæmorrhagic or softening character, almost uniformly cause a rise of temperature. 4. There is as yet no clinical evidence that lesions of the basal ganglia or the parts about them cause temperature rises on account of destruction of certain thermic centres; in other words, the clinical and pathological evidence of thermic centres in the human brain, aside from the parts mentioned, is yet inadequate. 5. Finally, gentlemen, I would specially impress upon you the great value, from a diagnostic point of view, of a careful study of the temperature changes after apoplectic strokes. The temperature should be observed on each side of the body, in the rectum, also, if possible. With data thus obtained one can, I feel sure, gain much more positive evidence as to the nature of the lesion in these cases, and I have repeatedly been able to satisfy myself, in my clinical work, of the nature of the lesion by means of the methods referred to. I do not believe that with the help of the numerous factors which we now have in aiding our diagnosis there are many cases of apoplexy in which it is difficult to make a diagnosis. The old-time tabulation of differential points in diagnosis between hæmorrhage and acute softening still remains of value. We need, and must use, all the helps possible; but if we, in addition to other methods, carefully apply the thermometric, I am sure we can reach vastly more satisfactory results. J. C.

**A Case of Subcortical Tumor Treated by Operation.**—Beever and Balance. (*Brit. Med. Journ.*, January 5, 1895.) The salient symptoms presented by the patient were as follows: First, the gradual onset of the paralysis, involving successively the right ankle, the knee and the hip, and then extending after the lapse of seven months to the joints of the right hand, and then to the whole upper extremity. Finally speech became affected. Second, the classical symptoms of intracranial pressure were present—headache, vomiting and optic neuritis. Third, the mental condition gradually deteriorated. Fourth, there was some loss of sensation affecting the right limbs chiefly while the face escaped entirely. Fifth, there was no family history of tubercle, and no personal history of tubercle or syphilis. Sixth, under anti-syphilitic remedies, taken for over six weeks, the patient's condition not only did not improve but grew worse.

The operation consisted of first a preliminary one, done six days before for exploration and removal of the tumor, which was found subcortically in the upper part of the motor region. Four months after removal of the subcortical tumor, which had been found to be sarcomatous innature and semi-gelatinous in consistency, the patient was examined and found to be restored to her normal mental condition; speech is per-

fect, there is no headache, and the movements of the face are normal. The right upper extremity semi-paretic, considerable rigidity at the joints, marked inability to extend the fingers and hand, considerable ataxia. Fingers kept straight, phalangeal joints extended, metacarpophalangeal flexed. Grasp, d. 65 R. 11. L. Frequent slight clonic spasms of first finger and thumb (no tonic spasm), which are increased by emotion. She can extend the fingers but not the thumb; can flex well the thumb and fingers; can extend wrist to line with forearm, but not beyond; can pronate and supinate forearm; can flex and extend elbow; can elevate humerus to right angle, but not beyond; no power to shrug the shoulders. In the right lower extremities she has not the least power in the toes and very little in the foot. The right knee she can extend powerfully, but flexion is weak; extension and flexion of the hip good, but weaker than the left; knee-jerk excessive. No anaesthesia, no loss of muscular sense.

A remarkable feature of the case is that, though so large an area of cortex was removed, the patient recovered sensation completely, and with the exception of the toes, the ankle and the shoulder, she has recovered almost as completely as regards motion, but with diminished strength as compared with the other side. The authors believe that this can be explained, according to the theory of Hughlings Jackson, as follows: That all movements of the upper limb, for instance, are represented in all parts of the upper limb area of the cortex, but in different degrees, and that it is not possible to completely paralyze the upper limb unless the whole of this area is removed. In their case some of the upper limb area remained.

J. C.

**Pseudo-Hypertrophic Paralysis in Late Life.**—Desterac. (*Congress de Médecine Intern. Bullet. Med.*, November, 1894.) The author describes a case of pseudo-hypertrophic paralysis in a patient sixty-seven years old. He remarks the absence of heredity, the existence of a cervical kyphosis, the absence of reaction of degeneration, the very late date of onset. He admits that it must be considered a primitive myopathy.

J. C.

**An Hysterical Form of Raynaud's Disease.**—Levi (*Archives de Neurologie*, Jan., 1895).

The author described a patient, 43 years old, who several years before had a severe attack of polyarticular rheumatism, and later, under the influence of chagrin and the emotions, developed neurasthenia, associated with suicidal ideas, which terminated in hysterical crises, and which in May, 1892, under the influence of a most severe moral shock, was the starting point of Raynaud's disease. The asphyxia of the extremities manifested itself with the characteristic intermittency and in the usual place. At the end of a certain time there was installed a true state of the disease with ten or twelve crises a day, each one of a duration of from one to two hours, and this was associated with aliguria and anuria. Hypnosis revealed the nature of the affection, and modified considerably the vaso motor neurosis and the urinary trouble. Under its influence the anuria was replaced by polyuria. The crises in the extremities were diminished in frequency and duration.

J. C.

**The Causation of Hemiatrophia Facialis Progressiva.**—Baerwald. (*Deutsche Zeitschr. f. Nervenheilk.*, vol. v., part 6.) A young, healthy man developed a progressive hemiatrophy of the face directly after a swelling and inflammation of the submaxillary gland which had been associated with angina. The author thinks it possible that the angina caused an infectious inflammation of the terminal branches of the fifth nerve, and that this would tally with the findings in Mendel's case, which were those of progressive peripheral neuritis of the trigeminus and secondary atrophy of the descending root of the trigeminus.

J. C.

**The "Anxiety Neurosis."**—Freud. Ueber die Berechtigung,

von der Neurasthenie einen bestimmten Symptomencomplex als "Angst-neurose" abzutrennen. (*Neurol. Centralb.*, January 15, 1895.)

Neuro-pathology, the author says, can only be a gainer by an attempt to separate from neurasthenia any group of nervous disturbances, the symptomatology of which, on the one hand, binds the members of the group closer to each other than to typical neurasthenia, and which, on the other hand, shows essential variations in etiology and mechanism from the ordinary neurasthenic group.

Freud proposes to separate such a group, to which he gives the name "anxiety neurosis." This neurosis may present a complete or incomplete picture, may occur alone or in combination with other neuroses, and is characterized by the following symptoms:

1. General irritability, although occurring at times in many states, is a constant symptom in the "anxiety neurosis," and is especially manifested as auditory hyperæsthesia, a hyper-sensitiveness to noise. This is frequently the cause of insomnia.
2. Anxious apprehension and the attributing of importance to trivial circumstances. This symptom shades off on the one hand into a not abnormal anxiety, and on the other into hypochondria; on another side it touches a hyper-conscientiousness which may develop into a veritable insanity of doubt.
3. An "anxious fit" may occur almost without any intervention of consciousness or ideation. It is then ordinarily accompanied by marked somatic signs, palpitation, perspiration, dyspnoea, bulimia and the like, and these may so overshadow the disturbances of the psychic state as to make them almost invisible.
4. Night terrors of adults.
5. Dizziness, varying in degree from slight giddiness to marked vertiginous attacks.
6. Two groups of "phobias": the first, the fear of serpents, storms, vermin, darkness, etc., embracing also super-scrupulousness and irresolution, is born of the chronic apprehensiveness; the second, agoraphobia and its congeners, takes its rise in the tendency to vertiginous attacks with anxiety.
7. Nausea, bulimia, diarrhoea and occasionally cystic tenesmus.
8. Various paræsthesiæ and a hyper-sensitiveness to pain.
9. Many of the symptoms that accompany or represent the "anxious attack" may become chronic, especially the dizziness, diarrhoea and paræsthesiæ. The etiology of this neurosis, according to the author, lies almost wholly in some abuse or deep impression in connection with the sexual function. An etiological classification would be:

For women—

1. Anxiety of adolescents, or virginal anxiety, caused in maturing girls by the sudden presentation or solution of the sexual problem.

2. Anxiety of the newly married, occurring in young wives who are at first anæsthetic to intercourse, but in whom the somatic processes of intercourse are complete.

3. Anxiety of wives whose husbands are not very potent or suffer from premature ejaculation.

4. Anxiety of those whose husbands indulge in incomplete coitus.

5. Anxiety of widows and of those who restrain the sexual impulse.

6. Anxiety of the menopause and final exaggeration of the sexual instinct.

For men, an analogous division—

1. Anxiety of the continent.

2. Anxiety from excessive sexual excitement with ungratified desire. This etiology produces the most typical cases.

3. Anxiety from indulgence in coitus interruptus.

4. Anxiety of men who pass through a climacteric with increased desire and diminished virility.

Two classes of cases include both sexes: 1. Subjects neurasthenic from masturbation become victims of the "anxiety neurosis" on relinquishing the vice; but in men this applies only to those who are still

potent. 2. The disease may be caused by overwork, worry, etc., without any sexual influence.

The relations of this neurosis with others are intimate; in the majority of cases it is combined with one of them, but in such cases the author insists upon the proposition that in every case of mixed neurosis several specific causes can be traced. Thus a woman always hysterical indulges in coitus reservatus, and has added to her hysteria the "anxiety neurosis;" a man, neurasthenic from masturbation, enters into unnatural sexual relations with one of the opposite sex and acquires in addition the corresponding specific neurosis. On the whole this symptom-group most closely resembles hysteria, but is more somatic in origin and mechanism.

PATRICK (Chicago).

**A Case of Acromegaly.**—By Linsmayer. (*Wien. Medic. Wochenschrift*, 294, 1894.) The case is that of a man, sixty years of age, in whom in the course of two years symptoms of acromegaly appeared. The enlargement of the affected extremities was due mainly to hypertrophy of the bones. Motility, intelligence and the organs of sense were intact. Speech was difficult. The patient succumbed to progressive cardiac paralysis. The autopsy showed, in addition to the enlargement of the extremities, hypertrophies of the internal organs, particularly of the heart. The genital organs were atrophied.

MEIROWITZ.

**Hæmatoma Auris.**—Middlemass and Robertson. (*Edin. Med. Jour.*, December, 1894.) There is strong evidence in favor of the contention that the proclivity of the insane to othæmatoma is due to a peculiar degeneration in the cartilage of the ear. This change is due to the same abnormal nutritional state which induces lesions of scalp, skull and dura mater to which the insane are specially prone. If this view is correct, then the seat of the hæmorrhage must be in the cartilage, though it will be apparently within the perichondrium, with which the new fibrous tissue which replaces the degenerated cartilage always blends. As the perichondrium normally merges into the cartilage by insensible gradation, one cannot accurately speak of the blood as lying beneath the perichondrium. It must be outside, in it, or in the cartilage. Tischkow believes that when this degenerative change is fully established rupture of the new vessels may occur quite spontaneously. Even if a slight traumatic element is added the risk of hæmorrhage will be greatly increased. It is very improbable that the disease is of a specific infective nature. The vital processes subsequent to the outpouring of the blood are merely those which occur after hæmorrhage into other similar situations. The formation of the cartilaginous nodules in the cicatricial tissue is easily understood if it is admitted that the whole or part of the perichondrium lies external to the blood effusion. The almost invariable occurrence of the tumor on the anterior aspect of the cartilage has never been satisfactorily explained.

FREEMAN.

**Nervous Dyspepsia.**—H. Moway, M.D. (*Medical Record*, January 5, 1895.) According to this author, much that is written on the subject of this disease is still to-day simply a jumble of the most varied states, including reflex gastric irritation, organic nervous disease, hysteria, neurasthenia and general intoxication of the system. Leyden has fixed the limit to this category on the basis that when we apply the term nervous to any particular malady we mean to describe a symptom complex which cannot be accounted for by anatomical changes. On this basis must be excluded from the group of nervous dyspepsia all those morbid conditions of the stomach which, though chiefly due to the nerves, are connected with anatomical processes in the nerves, or can with great probability be referred to such. Upon this ground must be excluded the gastric crises of tabes, gastric disturbances in pregnancy, the dyspepsias of anemia, etc. In neurasthenia and hysteria it is evident that the gastric symptoms are but part of these conditions and not the expression of an idiopathic affection of the stomach. He defines nervous

dyspepsia as an ailment of the stomach, without anatomico-pathological characteristics, dependent solely upon the nerves of the stomach. The stomach is primarily affected, and if any symptom on the part of the general nervous system appears it is due to an irritation proceeding from the stomach, and therefore a secondary manifestation. Treatment must be directed to the stomach. Among the most noted symptoms are: A thin, white coating to the tongue; lack of hunger, although when the patient begins to eat the food tastes good, but when he stops the misery begins; a sense of fullness in the stomach and eructations which give only temporary relief. But the most unpleasant symptom is a condition of general irritability. This feeling continues for an hour and a half to three hours, and disappears with the distress in the stomach. After these symptoms have abated, the patient experiences a craving feeling in the stomach, relieved by food, but again followed by distress and irritability. A little food, as a tablespoonful or two of milk, will allay this, with the advantage that no distress will follow. Among the causes he mentions, first, mental shock; second, tobacco smoking. The patient recovers quickly from the shock, he is not depressed, and his system would be at its normal were it not for his stomach. The cases caused by smoking must be excluded from those in which, from the large quantity of tobacco used, the system has become intoxicated and gastric disorder, as one of the manifestations of such intoxication, appears. Concerning the pathology he says there is nothing abnormal in the chemismus and the motor power of the stomach is not to any degree impaired. It can be assumed, however, that the sensory nerve of the stomach, the vagus, is in a state of hypersensitiveness which manifests itself upon the ingestion of food by the pressure thus produced upon the ultimate nerve-filaments. He also believes an inhibition of all muscular activity of the stomach occurs at once upon the close of a meal, due to a closure of the pylorus. Nervous dyspepsia is to be differentiated from the so-called *neurasthenia gastrica*. In the latter more or less of the characteristic features of neurasthenia are always present. Although the prognosis of nervous dyspepsia is regarded as unfavorable by many high authorities, the writer states that the disease as described here is very amenable to treatment, even in cases of long standing. The histories of six cases observed by the author are reported in which very brilliant and rapid results were obtained. Beyond hinting that strict dietary regulations are to be observed, the paper gives no details of the treatment carried out in the cases mentioned, which is very disappointing to the reader and much to be regretted.

FREEMAN.

**The Urine in General Paralysis.**—Klippel and Serveaux (*Archives de Neurologie*, November, 1894).

The conclusions of these investigators are:

The urine of general paralytics is essentially variable in the volume, quantity of urea, of phosphates, and of chlorides passed in twenty-four hours. While there is no absolutely constant variation in the great majority of the cases, there is a departure from normal. In the second stage of the disease there is no polyuria; the urine is of a low specific gravity, pale in color and somewhat turbid, with an abundant mucus deposit; while the excretion of urea is diminished, and the excretion of phosphates still more so, the chlorides are abundantly present. There is very frequently peptonuria, and the presence of acetone is nearly constant. Not infrequently albumin in very small quantities is found.

J. C.

**Syphilis Simulating Amyotrophic Lateral Sclerosis.**—Ballet (*Semaine Medicale*, November 21, 1894).

The author reports the case of a patient who had the local lesion of syphilis ten years previously and secondary symptoms three years before present attack: A man 34 years old, of neurotic temperament, noticed after a severe disappointment that he was depressed, introspec-

tive, bodily weak, and had lost flesh. A few months later he had a kind of fainting spell just before retiring, and this was followed the next day by difficulty in speech and partial inability to use the right side of the body, particularly the right leg. The arm continued to grow numb, and a week later it was completely paralyzed. After a few days a similar condition developed in the left side of the body, and the patient was quite unable to raise himself from the lying position or move any of the extremities. The speech difficulty increased, the face became expressionless, there was inability to close the lips, and saliva continually dribbled. The movements of the tongue were slow and very limited, the velum palati paralyzed, and the speech reduced to an unintelligible mumbling. Considerable difficulty in swallowing, especially for solids, and continuous attempts at swallowing provoked distressing attacks of suffocation, which were attended with cyanosis and impending asphyxia. The arms lay powerless by the side; the forearms semiflexed, the hands and fingers in the position of "claw" hand. The lower extremities were quite as powerless as the upper. Reflexes markedly exaggerated; extensive muscular atrophy particularly manifest in the thenar and hypothenar eminences, the interossei of the hand, the anterior muscles of the forearms, and the posterior extremities. Nutrition of the tongue normal. All the symptoms more exaggerated on right side of body.

Integrity of sphincters and absence of sensory symptoms. The patient was put onunctions of mercury and the internal administration of potassium iodide, and these were increased to the point of tolerancy. In less than a month after treatment was instituted the speech difficulty began to disappear, shortly afterward swallowing was less difficult, the wasting of the muscles was arrested and the strength of the patient began to increase. Three months later the patient was able to walk, the speech was greatly improved, the atrophy had nearly completely disappeared, but the patient continued highly emotional, nervous, and indulged in attacks of laughing and crying without any apparent cause.

In discussing the diagnosis the author inclined to the view that the disease was in reality a pseudo-bulbar paralysis of syphilitic origin. Multiple neuritis was excluded by the absence of sensory troubles and the presence of exaggerated knee and elbow jerks and the emotional symptoms. The suddenness and rapidity of the disease excluded amyotrophic lateral sclerosis. Theoretically, a medullo-myelitis causing a destruction of strands of fibres in the anterior pyramids and extending to the nuclei of the medulla and anterior cornu would account for the symptoms, but the author thinks the process was too diffuse to consider it an entirely motor affection, and believes that the lesion was a double central focus of disease involving certain fasciculi of the internal capsule and central gray nuclei in that neighborhood. J. C.

#### THERAPEUTICAL.

**Hot Baths in Protracted Cerebro-Spinal Meningitis.**—Aufrecht (*Die Therapie der Gegenwart*, January, 1895.) reports the case of a man twenty-five years old in whom at the end of the third week's illness benumbed sensorium, constant delirium and persistent opisthotonus were present. The fever had subsided, but the pulse remained rapid. Reasoning from previous experiences the prognosis was considered unfavorable. After the somnolence had lasted ten days it was decided on account of the low temperature and the frequent small pulse, to employ hot baths. The patient received from the 15th to the 17th of April twelve baths in all, at 40° C. and of ten minutes' duration. Even after the first baths a striking improvement was observed. Gradually the sensorium cleared up, pain and opisthotonus diminished, incontinence of urine and feces disappeared, the abducens nerve assumed its

normal function, and the power of speech returned. Upon discontinuing the treatment nocturnal headache and delirium reappeared, whereupon three more baths were given, after which all symptoms of disease vanished. Aufrecht reports this single observation to encourage the further trial of the very favorable effects of hot water.

FREEMAN

**Treatment of Tubercular Meningitis.**—By Dr. R. Hirschberg. (*Bull. Général de Thérapeutique*, November, 15, 1894.) According to the author, death in cases of tubercular meningitis is caused not by the development of the tubercles but by intracranial compression, by cerebral asphyxia. The rational treatment of the disease lies, therefore, in trephining and drainage, procedures similar to those employed with good results in tubercular peritonitis. The operation should, however, be performed before coma sets in. Ord and Waterhouse report a case of tubercular meningitis in a child five years old, in which trepanation of the occiput was done six weeks after the onset of the disease. The dura and arachnoid were divided, and a probe and drainage tube introduced between the cerebellum and the medulla oblongata. The drainage was followed by an amelioration of the severe symptoms. In spite of the fact that the tube was kept in situ for seventeen days, and an intercurrent attack of measles developed, the child got well.

MEIROWITZ.

**On the Analgesic Properties of Guayacol.**—By Aporti. (*Gaz. d. asp. Deutsche Med. Ztg.*) In a number of cases of pleuritis, pneumonia, gastralgia, acute articular rheumatism, myelitis, dysmenorrhœa, tubercular meningitis, neuralgia, neuritis, etc., the topical application of mixture of equal parts of glycerine and guayacol, two to three times daily, produced prompt and lasting effects. There are, however, contra indications for its use, as the presence of a high fever; nor is it always borne well, as cauterization and neurosis of the superficial structures of the skin may follow its application.

MEIROWITZ.

**The Operative Treatment of Spasmodic Torticollis.**—Richardson and Walton. (*American Jour. Med. Sci.*, January, 1895.) After reviewing the therapeutics of spasmodic torticollis the authors reach the following conclusions: 1. Palliative treatment, whether by drugs, apparatus or electricity, will rarely prove successful in well established spasmodic torticollis. 2. Massage may prove of value in comparatively recent cases. 3. Resection affords practically the only rational remedy. 4. Operation on the spinal accessory nerve may afford relief, even if other muscles than the sterno-cleido-mastoid are affected; on the other hand, the affection previously limited to the sterno-cleido-mastoid may spread to other muscles in spite of this operation. 5. No fear of disabling paralysis need deter us from recommending operation, as the head can be held erect even after the most extensive resection. The most common combination of spasm is that involving the sterno-mastoid on one side and the posterior rotators on the other, the head being held in the position of sterno-mastoid spasm with the addition of retraction through the greater power of the posterior rotators. 7. It seems advisable in most cases to give preference to the resection of the spinal accessory as the preliminary procedure.

J. C.

## Society Reports.

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### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, held at the New York Academy of Medicine,  
on Tuesday Evening, January 8, 1895.*

Dr. EDWARD D. FISHER, President, in the chair.

#### A CASE FOR DIAGNOSIS.

Dr. GREME M. HAMMOND, through the courtesy of Dr. M. J. Echeverria, presented a patient who exhibited an extraordinary condition of the muscular system. She is a girl thirteen years old and is one of a pair of twins. There is nothing of importance in her family history and no hereditary influence can be shown to account for her peculiar condition. She has always been well and singularly free from the ordinary diseases of childhood. About two years ago her family physician first observed the remarkable hardness of her muscles. This has gradually increased, until at the present time there is not a muscle of the body which is not almost as hard and unyielding to pressure as cartilage. This hardness is most pronounced in the muscles of the face and back of the neck, and in those of the thorax and arms. When the muscles are relaxed and at rest the hardness is less apparent. There is absolutely no hypertrophy, neither is there atrophy. The child seems to be well, strong and active. She is as agile as other children generally are, and jumps, walks and runs freely. Scleroderma is not present. The skin is loose over the muscles and is everywhere apparently well nourished. The freedom and quickness of movements readily distinguishes this condition from Thomson's disease. The reflexes and electrical reactions are normal.



A CASE OF ACUTE ARSENICAL POISONING,  
WITH SUBSEQUENT MULTIPLE NEURITIS.

BY DR. P. MEIROWITZ. (See page 178).

Dr. C. A. HERTER said he considered the case an interesting one because it is comparatively rare to see multiple neuritis produced by a single dose of arsenic; it is not so uncommon to see it produced by the injudicious use of arsenic given for medicinal purposes. The case is also interesting because it enables us to fix accurately the time when the influence of the drug on the peripheral nervous system was sufficiently pronounced to give rise to actual paralysis. This period, in the case narrated, was about two months. The speaker expressed the opinion that in many instances a multiple neuritis of toxic origin does not manifest itself until some time after the ingestion of the toxic substance has ceased. In one case coming under his observation, a woman suffering from alcoholism, a neuritis developed six weeks after the patient was admitted to the hospital, during which time she had practically no alcohol given her. Another point of interest is the manner in which the cerebral symptoms are produced in cases of arsenic or lead poisoning of long standing. In a case of the latter recently observed, an encephalopathy developed after a paralysis of all four extremities. Ordinarily this condition would be attributed to the action of the lead on the brain primarily, but as the man had a nephritis of long standing it was difficult to say, as it is in all these cases, whether the cerebral symptoms were due to the poison directly or were of uræmic origin.

Dr. MARY PUTNAM-JACOBI said that when we consider the enormous dose of arsenic which this boy took, and the comparatively small part of it which it seems he vomited afterwards, it appears probable that much of the poison must have been stored up in the liver to find its way later into the circulation in small quantities at a time. Such gradual secondary poisoning would account for the later symptoms, the neuritis. From the history of the case it seems almost incredible that so large a dose could have been taken without immediate fatal results through violent gastro-enteritis or from choleric-form collapse.

Dr. WILLIAM M. LESZYNSKY, in reply to Dr. Herter, said that in his experience cases of arsenical poisoning, due to the administration of the drug medicinally, are

not very frequent. He has seen five or six cases of multiple neuritis due to the ingestion of a single large dose of arsenic.

Dr. CAROT referred to a case reported in Boston where a child suffered from symptoms of arsenical poisoning, and it was found on investigation that the material of the nurse's dress contained a large amount of the drug.

The PRESIDENT, DR. FISHER, said that in these cases of metallic poisoning it is impossible to fix any limit as to the time when the signs of neuritis may come on, as the poison often remains stored up in the system indefinitely. This is shown in persons who have been exposed to lead, and who long afterwards may develop the symptoms of lead poisoning when potassium iodide is given them. In one case of acute mercurial poisoning coming under his observation, a neuritis developed within forty-eight hours after the ingestion of the poison.

### THREE CASES OF UNILATERAL LESION OF THE SPINAL CORD.

Presented by Dr. PEARCE BAILEY.—The first patient was a male, aged 47 years, a Norwegian. Soon after his arrival in this country, twelve years ago, he was stabbed in the back of the neck, close to the skull, on the left side. He was unconscious for three hours after the receipt of this injury, and was removed to the hospital. On regaining consciousness he found himself hemiplegic on the left side. The fact that he was hemianæsthetic on the right side was not noticed until some time later. His head hung limp upon his right shoulder, and for five weeks he was confined to bed with his head in supports. At the end of that time the power began to return to the leg, and still later to the arm. Complete return of muscular strength has not occurred in either, and though the leg is better than the arm, both are stiff. Sensations in these limbs remained normal. On the right side tactile sensibility was but slightly affected, while sensation for pain and temperature was so much impaired that ordinary cuts, bruises, etc., caused no pain unless very severe; he could hold a piece of ice in his right hand indefinitely, and could not distinguish between hot and cold water. A bed-sore developed over the right buttock, and there was temporary

paralysis of the bowels. He continued to improve for eight months, and since then his condition has remained unchanged. All muscles respond readily to mild faradic currents. The right knee-jerk is active and the left considerably exaggerated, and a clonus may be easily elicited on the left side. The man's motor symptoms are identical with those that might be furnished by a cerebral hemiplegia. As regards the localization of the injury in this case, Dr. Bailey said there was some difficulty in locating the exact segment implicated, because of the absence of recognizable injury to the anterior horn. It is probable, however, that the knife blade entered in the immediate region of the atlas and axis, and the area of anæsthesia indicates that the fourth cervical segment was the one injured.

The second patient presented by Dr. Bailey was also a case of stab wound in the neck, on the left side, followed by paralysis of the arm and leg on the corresponding side, with a loss of superficial reflexes and an exaggeration of the deep reflexes. There was no loss of muscular sense on either side. There was left myosis, with loss of the cilio-spinal reflex. On the right side there were analgesia and thermo-anæsthesia, and both the deep and superficial reflexes were present. In this case the sensory loss corresponded to the area supplied by the fifth cervical segment.

In the third case presented all the symptoms were confined to the legs and lower part of the abdomen. On the right side there was paresis of the leg, with slight contractures; the muscular sense was unimpaired; the knee-jerk was exaggerated; the cremasteric and abdominal reflexes were present; clonus was obtainable. On the left side there were analgesia and thermo-anæsthesia; tactile sensibility was not impaired; the cremasteric and abdominal reflexes were present; the knee-jerk was absent. The motor disturbances on the right side consist in weakness, with some stiffness of the leg. The gait is paretic rather than spastic. There is no hypertrophy nor atrophy, no fibrillary twitchings, no inco-ordination, no pain nor hyperæsthesia. There is some loss of bladder control, and some lessening of sexual power. The bowels are regular. Dr. Bailey said this case appears to be one of right unilateral gliosis of the lower dorsal region. In connection with his cases he exhibited a number of charts showing the location of the areas of anæsthesia.

Dr. HERTER referred to a case coming under his observation where the patient received a stab-wound on one side of the neck, which gave rise to motor symptoms on the corresponding side, and sensory symptoms—the exact character of which he cannot recall—on the opposite side. There was also myosis and the cilio-spinal reflex was lost. When the case was first seen the knee-jerks were absent on both sides, although the injury was high up in the cervical region. The loss of the knee-jerk on the side where the motor paralysis existed was persistent, while that on the opposite side returned after about ten days.

### TUMOR OF THE OPTIC THALMUS, WITH AUTOPSY.

By Dr. EDWARD D. FISHER.—The patient was a woman, aged 25 years; had always enjoyed good health. She had two children, both healthy. Her family history was negative, with the exception that her father died of so-called softening of the brain, and that in several members of the family there was exophthalmus, but no other symptoms of Graves's disease. During her first confinement there was marked enlargement of the thyroid gland, which, however, subsided. Following her second pregnancy, the thyroid again enlarged, which proved permanent; at no time, however, was there any rapidity of the pulse.

The patient first complained of severe headaches in April, 1894. At that time her eyes were examined, but no changes were observed in the optic discs. In June the pain in the head became excessive, especially in the morning, and was located on the right side and posteriorly. The patient was somewhat stupid. Her speech was not affected. The pupil of the left eye was dilated, but reacted normally to light. Her vision was unimpaired. There was slight left paresis of the face; the tongue did not deviate; taste was normal. The thyroid gland was enlarged on the right side. The left arm and hand showed marked weakness. There was pronounced ataxia. Sensation was much diminished and impaired as to touch and pain. She was unable to differentiate between heat and cold. The left lower extremity was not affected. There was no vomiting nor convulsions; the pulse varied from 65 to 70 per minute; the temperature between 97.5 and 98.5. Her condition continued to grow worse and the headaches persisted.

On July 21, 1894, when Dr. Fisher first saw the patient in consultation with Dr. Julius Rosenberg, there was marked paresis of the whole left side, with flaccidity and diminished reflexes. The sensory condition showed generally reduced response to all tests. The mental condition was somewhat affected, and the patient was hysterical; this, in conjunction with the flaccid paralysis with the complete dragging of the foot on walking, making a diagnosis of hysteria probable. The paralysis, headache and mental stupor gradually increased, and the sight began to fail. On August 31, an examination of the eyes made by Dr. Carl Koller revealed left hemianopsia and choked disc and paralysis of the left abducens. A diagnosis was then made of tumor at the base of the brain, involving the right crura cerebri above the third nerve, and compressing the right optic tract. On September 12 blindness was complete, with optic atrophy. The motor and sensory symptoms on the paralyzed side remained unchanged. The patient was subject to hallucinations of sight and hearing. A diagnosis of tumor involving the optic thalamus was now made. The patient went into a condition of coma and died on September 16. At the autopsy a large vascular tumor, the seat of a recent hæmorrhage, was found involving the right optic thalamus and compressing by its extension downwards the optic tract on that side; the growth proved to be a glioma. The special points of interest in this case were the flaccid paralysis, the diminished patellar reflex and the rapid onset of the eye symptoms, which were probably due to the rapid growth of the tumor towards the end.

#### TUMOR OF THE AQUEDUCT OF SYLVIVS, WITH PRESENTATION OF SPECIMEN.

By DR. JOSEPH COLLINS.—J. F., single, 18 years old, a plumber's assistant, was admitted to the Hospital for Nervous Diseases on October 6, 1894. When first admitted he stated that his illness dated from a fall, which he received about six months before. He afterwards confessed that he had not been well for two years, when he began to have dull, sleepy feelings, which his people attributed to laziness. He took no interest in his work and it was with difficulty that he was able to keep his place, although he did so until four months before coming to the hospital. During these two years he had

attacks of dizziness, some headache, and occasional weak spells, but he had never lost consciousness. He continued to grow worse, and in July, 1894, he lost control of the bladder and became uncertain and somewhat staggering in gait.

On the patient's admission to the hospital he was examined with the following results: He was a large, dull, overgrown-looking boy, with an expressionless face. If left undisturbed he sits in one position the greater part of the time, and shows very little interest in his surroundings. He understands perfectly everything that is said to him, and answers intelligently but slowly. His memory is fairly good. He is deficient in mental power and apprehension, and continuous mental exertion is beyond him. His gait is the characteristic inco-ordination of one very drunk. When he attempts to walk he staggers, reels, then plunges, but saves himself before falling. The staggering is not confined either to the right or left. The patellar reflexes are normal. Sensation is unimpaired but not very acute. Vision good; the pupils react to light and accommodation. The ophthalmoscope shows choked discs, but not very marked. There is no ocular paresis nor paralysis. The hearing is good. There is no defect of smell or speech. The patient's temperature was continually subnormal, and he complained that he could not get thoroughly warm. He complained of spells of severe headache. He frequently passed his urine in bed or in his clothing, but this seemed to be due not so much to paralysis of the sphincter as to a lack of interest in responding to the calls of nature. There was no nausea or vomiting. There was no loss of flesh.

The patient's condition gradually grew worse, and on the morning of November 2, 1894, he was found dead in bed. The autopsy was made eight hours after death. The occipito-mental circumference of the head was  $25\frac{3}{4}$  inches; the occipito-frontal  $25\frac{1}{4}$  inches. There were no evidences of meningitis. On cutting off the cerebellum, medulla, pons and cerebral peduncles there was found a grayish, translucent mass filling the aqueduct of Sylvius, and projecting backwards like a tongue. Anteriorly, it reached as far as the splenium of the corpus callosum, and posterior commissure. The walls of the third ventricle were nearly destroyed, so that the third ventricle and the lateral ventricle practically formed one cavity. After hardening the brain in alcohol, it was seen that the

tumor extended downward almost to the upper termination of the fourth ventricle. Cultures of the tumor for tubercle bacilli were made with negative results. Drs. Brooks and Schmitt, who made the microscopical examination, expressed the opinion that the growth is tuberculous. In connection with his paper, Dr. Collins exhibited the specimen and a number of microscopical slides.

Dr. C. L. DANA referred to somnolence as a very important symptom in the diagnosis of tumors in the central part of the brain. In a discussion of this subject before the American Neurological Association some years ago, in connection with a case reported by Dr. Sinkler, the opinion was quite generally expressed that in cases where the growth is located in the deeper portion of the brain, not in any particular locality, however, somnolence and hebetude are much more pronounced than in cases where the growth is situated near the surface.

In reply to a question, Dr. FISHER stated that the patellar reflex in his case was lost on one side only.

Dr. HERTER said he regarded this as a rather remarkable feature of the case. In cerebellar lesions it is not uncommon to find one or both of the patellar reflexes absent, but with tumors of the brain in this region it is uncommon.

Dr. LESZYNSKY suggested that the loss of the reflex might have been due to inhibition. In a case of traumatic meningitis of the convexity of the brain recently under his observation, in a child eight years old, there was complete temporary loss of reflexes on both sides. This also occurred in a case of meningitis subsequent to pneumonia.

## Book Reviews.

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PRACTICAL URINALYSIS AND URINARY DIAGNOSIS. By Charles W. Purdy, M. D. The F. A. Davis Company, Publishers, 1894.

"To the professors, past and present; to the fellows, alumni, and students of my alma mater, the following pages are affectionately inscribed by the author." Such is the dedication of a book of 357 pages, claiming to be a practical guide for chemical and microscopical analysis of the urine. The dedication is unquestionably pompous, though one cannot help wondering how the "past professors" of the alma mater can enjoy it. "Over twenty-five years' experience, coupled with a somewhat liberal examination of the literature of this subject, as well as considerable practical observation and experiment, have enabled the author to contribute some original matter and methods, which he trusts will prove to be an advance in certain practical departments of this subject." This sentence strikes us in the preface, for it correctly places the standard of the whole work. Indeed, there is "some original matter" and a "liberal examination of the literature"—rather a liberal perusal.

The book is, no doubt, a good one; though not exactly practical in the sense of the author. He is too lavish in the perusal of the literature, and brings a good deal of matter that the practitioner must consider burdensome. The book is too much of a compilation, mainly from German authors, from whom the majority of the illustrations are borrowed. Among these, again, Peyer is the favorite author.

On page 71 the highly important "detection of albumin in the urine" begins. The first method is described under the title of "heat." Indeed, this method is the best even in our days; but if we use nitric acid for discrimination between phosphates and albumin, we are liable to be mistaken; especially if only small quantities of the albumin be present, since the slightest surplus of the acid will dissolve the albumin. All that is needed is a few drops of acetic acid (equal parts of a saturated solution of glacial acetic acid and water), which will not affect even slight traces of albumin. This method has proven the best in our hands, as well as in those of practitioners generally. Nitric acid should never be used. The author recommends a method with a solution of chloride of sodium and one or two drops of strong acetic acid. This method is superfluous, if we use dilute acetic acid. Only highly alkaline urine needs the addition of dilute acetic acid, in order to obtain the albumin test by boiling. The above mentioned simple test for albumin renders most of the eighteen methods described by the author superfluous. He correctly remarks on page 78 that he has, from long experience, come to be somewhat suspicious of tests for albumin which require previous acidification of the urine when searching for minute traces of albumin.

On page 101 another important chapter is dealt upon, viz., the "detection of sugar in the urine." Six different tests are described, all good, no doubt, but far too complicated. The simplest test (Moore's), mixing two-thirds of the urine with one-third of a ten per cent. solution of caustic potash with the characteristic color-changes by heat from



canary yellow to dark brown and black, is not even mentioned. Still it will yield satisfactory results, even in approximating the quantity of grape sugar. It is only exceptionally that we must resort to the fermentation test. Einhorn's small apparatus proved to be rather serviceable, though the author objects to it.

On page 159 we find the following remark: "The smaller crystals (of ammonium urate) often closely resemble those of sodium urate, and by some they are claimed to be identical." In the first place, neither sodium urate nor ammonium urate are a crystalline deposit being amorphous, granular and globular; in the second place, the small dumb bells of ammonium urate, as correctly depicted by Peyer, are formations in *statu nascenti* and cannot be mistaken for sodium urate. The latter is acid, the former alkaline.

On page 160 the author describes the forms of calcium oxalate. He correctly states that the so-called "dumb-bell crystals," which are no crystals at all, are merely the edge views of ovoid or circular discs with depressed centres. He omits to mention the concentric striations which every good lens shows upon the discs and brings the rather poor illustration of Peyer, likewise deficient in striations.

On page 166, speaking of the deposit of calcium phosphate and triple phosphates, he says that the urine, holding these salts, effervesces upon the addition of an acid. This is a mistake. Effervescence is due either to ammonium carbonate, always kept in solution and never seen in the deposit or to calcium carbonate. The latter salt producing small prisms is not even mentioned. Still it is of importance, because, unless due to drinking certain mineral waters, it invariably indicates wasting diseases of the osseous system, rhachitis, caries, otitis, etc., in which the lime salts are dissolved out and appear in the urine. On page 221 the author, however, mentions calcium carbonate concretions.

The chapters on "Hæmaturia" and "Pyuria" cannot be called good. Obviously, the microscope is called upon to determine the presence or absence of blood, pus corpuscles and epithelium. The latter will, in the majority of the cases, positively fix the source of the hæmorrhage or of the inflammation, which invariably is accompanied by pus-corpuscles, the products of the epithelia. The study of the epithelia is, therefore, of the utmost importance in microscopical urine analysis. If we look at figure 22, as illustration of epithelia after v. Jaksch, we can rest satisfied that with such a poor knowledge of the epithelia of the genito-urinary tract, no positive diagnosis of the locality of an inflammatory process, or any disease, is possible. The reviewer, who for twenty years has examined thousands of samples of urine, cannot but be astonished at the deficiency of these chapters. The author has, rather unjustly, ignored everything that was done in this line for the last score of years in our country. Why rely upon Germans, if so much original work was done here?

If the author makes the statement on page 294, that the acute interstitial nephritis has been described under several names, viz., suppurative nephritis, acute interstitial nephritis, pyelo-nephritis and surgical kidney, he simply proves that he is not conversant with the pathology of the kidneys and the diagnosis of their diseases from microscopical analysis of the urine. The headings above named mean widely different conditions. We cannot be surprised if, in the description of benign vesical growths, he omits the all-important shreds of fibrous connective tissue. If he says on page 309 that the "washed out" bits of tissue have no diagnostic value, since both malignant and benign growths of the bladder may prevent papillary surfaces of practically the same microscopical appearance, he is completely mistaken, because the appearance of such shreds is widely different in the instances mentioned. The second portion of the book, dealing with microscopical examination of the urine is decidedly inferior to the first part, mainly chemical in nature. At present we do

not hesitate to call the book a good one, as far as chemical analysis goes. This, however, is the least important for the practitioner, who lacks time for minute chemical examinations. What every practitioner ought to know, is the microscopical analysis. In order to render the book a perfect one, a thorough study of the pathology of the genito-urinary organs would be required. Thus the author would be enabled to tell us much more about cystitis, pyelitis and nephritis. He would be able to diagnosticate from the microscopical analysis of the urine, a number of diseases, to which, at present, no allusion is made; for instance, enlarged prostate, spermato-cystitis, masturbation, vaginitis, endometritis, sarcoma of the bladder, of the kidneys, etc.

C. HEITZMAN.

DIAGNOSTIC NEUROLOGY.—By Alexander B. Shaw, M.D.  
Pp. 114. Illustrated. St. Louis, 1894.

There is but little doubt that a work on the diagnosis of nervous diseases, if couched in simple and comprehensive English, would be well received by the general practitioner. While we are in sympathy with the author's intention, we cannot but regret that carelessness in applying the rules of syntax and in the construction of sentences have marred a work which might otherwise deserve a fair share of praise. Were these errors trivial or isolated it would not be worth while referring to them, but they occur with such surprising frequency and startling *abandon* that even the most liberal minded critic could not disregard them. An instance of what is meant is supplied in the paragraph on "Spastic or Tetanoid Paraplegia." The initial sentence supplies the startling information that "in well-developed cases the knees are often unnaturally approximated while seated." We neither admit the possibility of "knees being seated" nor that the author intended to convey any such meaning. Again, in the same paragraph we are informed "that the knees and feet are disposed to become interlocked, often crossing each other in walking." This is a feat that would floor a professional contortionist. It is unnecessary to quote further. The whole book teems with sentences of careless, and often of grotesque construction, which could not avoid seriously detracting from the value of a work of much greater pretension than the one under consideration. Most of the illustrations are greatly inferior to those which adorn the pages of other recent volumes. They are, in the main, reproductions of very amateurish photographs. Probably future editions, freed from the objectionable features which tarnish this one, will deserve only words of praise.

GRÈME M. HAMMOND.

THE  
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**Nervous and Mental Disease.**

**Original Articles.**

MYXEDEMA AND EXOPHTHALMIC GOITRE IN  
SISTERS, WITH REMARKS ON THE SYM-  
PTOMATOLOGY OF THE LATTER DISEASE.

BY A. R. OPPENHEIMER, M.D.

THE occurrence of exophthalmic goitre and myxedema in members of the same family has been observed by Arthur Maude,<sup>2</sup> whose cases, as far as I can ascertain, are the only ones on record. The following cases have recently been under observation at the Medical Clinic, and Prof. Osler has kindly asked me to report them:

CASE I.—Miss A., aged 19, admitted January 26, 1894, complaining of goitre and great nervousness.

*Family History.*—The father living and healthy; several brothers living and healthy; her mother is dead; cause unknown. There is no history of tuberculosis, carcinoma, or nervous diseases. She thinks that several members of her family have had "Bright's disease." Her sister is Mrs. B., Case II.

*Personal History.*—She had measles and scarlet fever when young. The catamenia have never been very regular, and have appeared only once in the last year.

The present illness began about three years ago. After a severe fright she became very nervous, and has been easily excited ever since. The heart has been

<sup>1</sup> From Prof. Osler's Medical Clinic Johns-Hopkins Hospital.

<sup>2</sup> Maude, St. Bartholomew Hospital Report, 1893.

beating very rapidly, and for about eighteen months enlargement of the thyroid and exophthalmos have been noticed. The voice has been husky of late; the hands tremble, especially on excitement.

The appetite is good; there is no nausea or vomiting; the bowels are regular. There is no cough or pain in chest, but she has attacks of dyspnoea.

*Status Præsens*.—Fairly nourished, slightly built, face markedly flushed, numerous areas of transient flushing on skin. Lips and mucous membranes of good color; tongue clean.

Marked double exophthalmos; no v. Græfe's sign.

Thyroid gland much enlarged symmetrically.

Pulse very rapid, varying from 100 to 140.

Lungs and abdomen negative.

*Heart*.—Apex heat in sixth space, very powerful and heaving; the sounds are loud, first is booming, second both loud and clear; no murmurs.

Fine tremor of fingers.

Urine negative; no sugar.

The patient was given the dried thyroid extract, gr. v. t. i. d. during her entire stay at the hospital—fourteen days. She neither improved nor became worse, but lost two and a half pounds in weight; pulse remained about the same. She left the hospital on February 9, 1894.

Her physician, Dr. H. B. Melvin, writes that she had to discontinue the thyroid, because of the increase of nervousness. She has been taking tinct. belladonna m xv., t. i. d. for two months, and has improved somewhat. In the last six weeks she has taken it very irregularly. On November 22, 1894, the goitre was much smaller, and the exophthalmos less marked. She is, however, very irritable and does not obey orders.

CASE II.—Mrs. B., sister of Miss A., aged 37, multipara.

*Personal History*.—She has always been healthy; has never had rheumatism or chorea.

Present illness began about three years ago, after nursing two children with measles, when she was pregnant. Some three months after birth of the child, she noticed that about two days before the menstrual period, the whole body would become more or less swollen, though never pitting on pressure; this would disappear when the menstrual flow began. During the past year the menstrual periods have appeared at about six to eight weeks interval, and the swelling has always been

more or less present, though worse at sometimes than others. The swelling never pits on pressure. Lately she has noticed that the skin has become dry, harsh and rough. She has also lost some hair lately. She always feels chilly. The appetite is good; the bowels are constipated. She is very talkative.

*Status Præsens.*—A woman of moderate height, but giving the impression of being very large. The face has a heavy, dull look; the cheeks and neck are very full, almost œdematous in appearance. The face is somewhat expressionless, and complexion has rather a doughy character. The supraclavicular spaces are markedly full, though there is no distinct puffiness. The arms and legs are decidedly swollen, though not pitting anywhere, the skin having a resistant feel and being everywhere dry, and in places showing a scaling of the epidermis. The finger nails are thin and show marked longitudinal striations; edges are slightly everted and show irregularities. The hair is dry and coarse.

The thyroid gland could not be felt; owing to the thickness of the neck the palpation was unsatisfactory.

The abdominal and thoracic viscera are negative.

The patient was given thyroid, gr. v., t. i. d., which in a week was reduced to gr. v., b. d. She at once began to improve. Her weight while in the hospital—fourteen days—went down from 189 to 178.5 pounds, and in the twenty-five days after leaving the hospital she lost almost as many pounds. Her pulse on admission was 60 to 70, and on discharge it was 80 to 100. She looked much brighter and felt better.

Her physician, Dr. Melvin, writes November 22, 1894, that she has steadily improved; she is now taking only three grains every other day. She is eight months pregnant, and it is interesting that, while in previous pregnancies she was very nauseated, this symptom is absent in the present one. The amount of the thyroid extract could not be decreased during pregnancy.

These cases are of especial interest in the bearing on the question of the pathogenesis of exophthalmic goitre. Although the disease has been well known for fifty years, and studied with especial care of late, there is still much difference of opinion as to its cause. The symptoms, for which any theory must account, have become more and more detailed, and are increasing constantly, until now exophthalmic goitre has one of the most complex symptomatologies.

The disease occurs much oftener<sup>1</sup> in women than in men, probably about twelve to one, though the estimates vary. It occurs oftenest in early and middle adult life, but cases have been reported in children and in persons over sixty years of age.

Many cases of heredity are in the literature. Hector Mackenzie<sup>2</sup> in forty cases, had five in whose family there was exophthalmic goitre. In Bull's case a mother and two sisters were affected. Oesterreicher reports the most remarkable instance; an hysterical woman had ten children, eight of whom had exophthalmic goitre; one of these children had four daughters with exophthalmic goitre. Arthur Maude<sup>3</sup> reports a case of a woman with myxœdema, whose daughter had exophthalmic goitre.

In addition there is frequently a neuropathic family history. The English have laid stress on rheumatism and quinsy.

The immediate cause may be some violent emotion, such as in Case I., but in many cases the history is negative.

There is the well-known classical triad of symptoms; exophthalmos, goitre, and tachycardia. In connection with the exophthalmos there are certain changes in the motions of the lids; v. Graefe's sign, inability of the upper lid to follow the downward movement of the eyeball. This sign is much less common than is generally supposed. It was not present in one of the hospital cases, seventeen in number. On the other hand, Sharkey<sup>4</sup> found it in about two per cent. of 613 cases of other diseases, and maintained it could be produced by steady staring in a perfectly normal person. Stellwag's sign (observed already in 1849 by White Cooper) is a widening of the palpebral fissure, owing to spasmodic retraction of the upper lid. In connection with this the patient winks less frequently than normally. This is much more constant than v. Graefe's sign. Occasionally retraction of the lower lid is seen, and still more rarely ptosis.

The ocular muscles are at times involved. Möbius's sign consists of a lack of convergence of the two eyes. Not only do they not converge on a close object, he says, but often one eye turns out instead of in. He believes this is only a symptom of the general weakness, as convergence requires more nervous strain than other co-or-

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<sup>1</sup> Mackenzie, *Lancet*, 1890, ii., 545.

<sup>2</sup> Sharkey, *Lancet*, 1890, ii., 877.

minated movements. External ophthalmoplegia has been often noted, as well as other affections of the ocular muscles.

A fourth cardinal symptom has been added to the classical triad—the tremor. It was described in detail and given its full clinical value by Pierre Marie. It is involuntary, fine, and rapid (eight to the second). It oftenest affects the hands and arms, but at times the head and lower limbs. It may become coarse and violent, but in those cases hysteria is always to be suspected. It is one of the most constant symptoms, and is of great value in the diagnosis of the “masked” cases (*formes frustes*).

The appetite varies from anorexia to bulimia, and is at times perverted. Vomiting is at times a most distressing and often fatal symptom. It may be pernicious, and post-mortem, no gastric condition to account for it is to be found. There is often a paroxysmal diarrhoea, a true intestinal crisis, aptly termed “bowel hurry” by Hingston Fox. It lasts but twenty-four to forty-eight hours, and then disappears for a time. It may, however, be constant. In one of our cases it was of the “morning diarrhoea” type of Lauder Brunton; four or five loose stools soon after breakfast, and no more for the rest of the day.

There is usually polypnœa, but no dyspnœa, unless from pressure by the goitre. Associated with this is a short, dry cough, and occasionally hæmoptysis. This, with the emaciation seen in exophthalmic goitre, has often given rise to the diagnosis of tuberculosis. Dr. Bryson has called attention to diminished chest expansion, but this has not been generally confirmed.

The voice often becomes high-pitched and nasal. In some acute cases (Mueller and Koeppen) it was nasal, monotonous, and very weak.

There is polyuria, and occasionally glycosuria and albuminuria. Boinet and Silbert\* have isolated three ptomaines from the urine, which, injected into animals, caused convulsive symptoms, but nothing like exophthalmic goitre. Gilles de la Tourette has found the usual constituents unchanged.

Fever is often present, usually only slight, but may be very high (Bertoye). It is interesting that in this fever the urine remains unchanged.

The skin is usually moist and reacts quickly; in one

\* Boinet and Silbert, *Rev. de Med.*, January, 1892.

case the prick of a pin caused a general sweat. Profuse sweats and subjective flushes are common. Vigouroux has shown that the electrical resistance is decreased. This has been confirmed, especially by Eulenburg and Kahler, but it is also present in neurotic subjects without exophthalmic goitre.

There is often some pigmentation of the skin. It varies from a dirty grey to a mulatto brown (in Oppenheim's case). Burton<sup>7</sup> remarked that the deep pigmentation is most marked at pressure points, as at the waist, under garters, under a ring, etc. The pigmentation may be so intense as to simulate Addison's disease.

Other skin affections have been observed: vitiligo, eczemas, urticaria, scleroderma, jaundice.

There are nervous symptoms aside from the tumor. Paresis of almost every muscle has been seen. Especially common is the paraparesis of the legs (the "giving way of the legs"), on which Charcot laid great stress. Atrophy of some muscles has occurred. Muscular cramps are not uncommon, and Maude and Chvostek have called attention to choreiform movements. Joffroy<sup>8</sup> has lately brought out a sign—paresis of the frontalis. The patient is requested to look down, and then, without raising the head, is to look at the ceiling. A normal individual will wrinkle his forehead, but in exophthalmic goitre the forehead remains smooth. This is not present in all cases and is to be seen in some hysterical patients.

Undoubtedly many of these pareses are hysterical, but according to Joffroy there is a true "Basedowian paresis."

Edema has been especially noted by Moebius<sup>9</sup> and Maude. It does not necessarily occur in dependent portions; in a case of Moebius's it increased from below upwards, being most marked just below the umbilicus. It often comes on suddenly and resembles angioneurotic edema.

Non-pitting, hard, œdematous areas have also been seen. Hektoen, of Chicago, lately had a case in which there was a solitary patch of this below the knee.

In a few cases (Sollier, Kowalewsky, v. Jaksch), there

<sup>6</sup> Oppenheim, *Berlin Klin. Wochenschr.*, May 7, 1888.

<sup>7</sup> Burton, *Lancet*, 1888, ii., 573.

<sup>8</sup> Joffroy, *Progrès Méd.*, December 23, 1893; January 27th, March 24th, March 31, 1894.

<sup>9</sup> Moebius, *Schmidt's Jahrbucher*, cccxx., 185.



has been true myxœdema associated with exophthalmic goitre, so that the above may be a transition stage.

The mental condition is nearly always changed, generally in the direction of exaltation. From mere irritability and "nervousness" up to a violent mania, all gradations have been observed. Melancholia, *delire de toucher*, arithmomania, and various delusions are occasionally present.

Exophthalmic goitre has been associated with various diseases of the nervous system: *tabes dorsalis*, epilepsy, paralysis agitans, hereditary chorea, syringomyelia, but oftenest with hysteria. Diabetes is occasionally present, though a pure glycosuria is much more frequent. These all seem to be more concomitants than true complications, and when autopsies have been made the lesions of the nervous disease were, apparently, unmodified by the exophthalmic goitre.

There is often great emaciation, entirely disproportionate to any apparent cause. It may be as marked as that of carcinoma, and, indeed, the disease has been called *cachexia exophthalmica*. In many cases, however, the patient is rosy and well nourished.

The disease may be incomplete, one or more cardinal symptoms being absent. These are the masked forms, the "*formes frustes*." Most frequently it runs a slow, progressive course. More rarely it is acute, and rapidly fatal. F. Mueller reported some very interesting, acute cases in 1893.

To account for these various symptoms, numerous theories have been advanced. Only three are, at present, well supported: 1. That it is a pure neurosis. 2. That it is due to a central (medullary) lesion. 3. That it is due to an increased, perhaps perverted function of the thyroid gland.

The arguments in favor of the neurosis theory are these:

1. The frequent neuropathic family and personal history.

2. The onset with emotion. H. Mackenzie especially calls attention to the facies being but an exaggeration of the physiological facies of fright; protuding eyes, flushed face, tremor, palpitation, and swelling of the throat.

3. The frequent association with hysteria, epilepsy and chorea.

4. The absence of any lesion to account for it.

5. The cases cured by operation on the nose. These

are reported by Hopmann, Hack and Fränkel. A minor operation on the nose has been followed by amelioration or disappearance of the symptoms. The very small number of cases precludes any definite conclusion. Many cases have had their nasal passages treated without any result whatsoever on the disease.

Against these may be urged that the meagre pathological testimony may be due to lack of skill. (Moebius remarks that in strychnia poisoning there is no definite lesion). The mortality is too high and the acute cases totally unlike a pure neurosis. Again, a patient's statements as to the cause of onset are always to be looked on with suspicion. It is notorious how frequently anterior poliomyelitis is attributed to a fall, and chorea to a fright.

The theory of a central lesion has been supported by Mendel, Hale White and others. Mendel's pupil, Mannheim, in his work on exophthalmic goitre, sums up the arguments in favor of this as follows: He first premises that all the symptoms could be explained by bulbar lesions. Besides this there are, pointing to the medulla as the seat of the disease.

1. The severe course of exophthalmic goitre, and its combination with other spinal cord diseases and with diabetes.

2. The experiments. This refers to Filehne's, Durdufi's, and Bienfalt's work. Filehne incised the grey matter of the upper part of the restiform body and had exophthalmos and tachycardia. On electrical stimulation he also had goitre at times. Durdufi repeated this, and incised also the tub. acustica, getting Stellwag's sign. Bienfalt practically confirmed Filehne's experiments.

3. The pathological anatomy. Several observers have found more or less hæmorrhage into the floor of the fourth ventricle. Hale White noted an old superficial hæmorrhage extending from the middle line to the corpus restiforme, and involving the sixth nucleus on one side. Mendel found one restiform body smaller than the other, and the right solitary fasciculus atrophied. At the time Mendel's paper was presented Oppenheim remarked that the corpora restiforma are normally so unlike, that a slight difference in size would have but little weight.

In most autopsies, however, the central nervous system has been negative, and in Mueller's acute cases only

slight, recent hæmorrhages were seen. Mannheim explains this by our poor methods of demonstration.

Finally we have the thyroid theory, led by Mœbius, in Germany, Joffroy, in France, and Greenfield and Byrom Bramwell, in Scotland. The arguments for this may be stated as follows :

1. Morbid anatomy. In all autopsies some changes have been observed in the thyroid gland, and frequently these have been in the direction of functional hyperplasia. In some cases, however, (notably Hektoen's) the gland was almost completely fibrous.

2. The action of thyroid extract. Soon after the discovery of thyroid therapy for myxœdema, Murray, and later others, warned against the effects of overdosage. These were, notably, tachycardia, tremor, headache, sweating, prostration and anorexia, diarrhœa and polyuria. In the *Soc. des Hôp.*, on October 12, 1894, Beclère reported a case in which exophthalmos developed after an overdose of thyroid. Finally, in the *Soc. des Hôp.*, on November 10, 1894, Ballet and Enriguez report experiments on a dog, injecting thyroid extract. The dog had two courses, and in each developed goitre, which diminished and disappeared when the injections were omitted.

3. The usual effect of thyroid administration in exophthalmic goitre is to increase the symptoms. There are exceptions to this, however.

4. The most successful line of treatment, so far, is that tending to diminish the bulk of the goitre. Out of sixty-eight operations on record, eighteen completely recovered ; in twenty-six there was more or less improvement ; nine showed no change ; in five death was almost immediate,<sup>10</sup> and in four death occurred within twenty-four hours. In four cases there was apparent cure, but the symptoms returned, and in two cases the operation was followed by tetany. This makes about fifty per cent. improved by the operation, and a mortality of about fifteen per cent. This supposes that all these cases reported were true exophthalmic goitre, which has been questioned by several writers.

5. The striking contrast of symptoms between exophthalmic goitre and myxœdema, as pointed out by Mœbius. This is well shown in the two sisters whose histories were given above.

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<sup>10</sup> One of these fatal cases was in this hospital.

	Miss A., <i>æt.</i> 19.		Mrs. E., <i>æt.</i> 37.
Pulse,	100 to 120.		58 to 70.
Heart,	Impulse heaving; apex in sixth; first sound loud.		Negative.
Eyes,	Prominent (no v. Graefe's sign).		Not prominent.
Thyroid,	Symmetrically en- larged.		Not to be made out
Tremor,	Present.		Absent.
Skin,	Moist; sweats.		Dry and harsh.
Face,	Flushed; red patches on cheeks.		Heavy, full, uniform color.
Tempera- ment,	Emotional and neu- rotic.		Apathetic and heavy.
Weight,	137.		189.
Thyroid treat- ment,	Had to be omitted because of increase in symptoms.		Resulted in steady im- provement.

6. Finally, the course of the disease is more like an intoxication. It is probable that the chief brunt of the toxæmia falls on the central nervous system, and this would give rise to medullary symptoms without marked lesions.

The question might possibly be cleared up by injecting animals with extract of thyroid from an exophthalmic goitre subject. This disease has been observed once in a cow, in a horse, and in a dog, so that they are susceptible to it.

## CIRCULAR INSANITY.—REPORT OF THREE CASES.

BY WILLIAM F. DREWRY, M.D., of Petersburg, Va.,

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CIRCULAR insanity (*alternating insanity, cyclothymia, folie circulaire; folie à double forme*) is a psychosis of such rarity I am induced to put upon record a report of three cases that have come under my personal observation. Reports of very few cases have appeared in the medical journals. Some systematic writers regard it as a mere subdivision of periodic insanity (Spitzka.) A distinguished alienist and author, of Scotland, however, has given us an admirable lecture on the subject. He says: "I have had under my care altogether about forty cases of typical *folie circulaire*." In the asylum at Morningside there were, says Dr. Clouston, in 800 patients sixteen cases of this peculiar form of mental disease. Dr. Spitzka, who was, I think, the first American to describe it, found in 2,300 cases of pauper insane four per cent. to be periodical, and its sub group circular insanity. Dr. Stearns states that less than one-fourth of one per cent. of cases in the Hartford (Conn.) Retreat classed as mania and melancholia have proved to be *folie circulaire*. Upon examination of the annual reports of the superintendents of hospitals for the insane in this country, I find in only a few reference made to this as a distinct form of insanity. In the New York State hospitals there is a regular uniform classification of mental diseases in which "circular (alternating) insanity" occupies a place. In the report of the Buffalo Hospital for 1892, in statistical table No. 4, "showing forms of insanity in those admitted, etc., since 1888," out of 1,428 cases, only one was "alternating (circular) insanity." In the St. Lawrence Hospital only one case in 1902 was credited to this special class. In the institution in Philadelphia, of which Dr. Chapin is the superintendent, 10,379 patients have been treated, only three of whom were diagnosed cases of *mania circulaire*. Of

the 900 cases of insanity in the State Hospital at Danville, Penn., less than four per cent. were put in this special class. There are in the Central (Va.) State Hospital (which is exclusively for the colored insane) 775 patients, three of whom are genuine cases of circular insanity, but they are included in "periodical insanity." This same custom evidently prevails in many of the other hospitals for the insane.

Periodical phenomena are seen everywhere in nature. This universal law of periodicity applies to insanity in general, as has been observed by those who come much in contact with insane people. Remissions and intermissions in diseases of the mind are familiar to us all. In his charming clinical lectures on mental diseases Clouston says: "One of the most fundamental of the laws that govern the higher functions of the nervous centres in all vertebrates is that of alternation and periodicity of activity and inactivity."

Between the ordinary simple variety of recurrent or periodic insanity and the strange type of mental disease under consideration, there are distinct differences. In the one, apparent rational periods, varying in length, intervene between attacks of mental disturbance, which, in each case, are similar in character. For instance, there may be a recurrent mania or a recurrent melancholia, etc. In the other (circular insanity), there is a rhythmic alternation in uniform order, in each particular case, of mental states from depression to exaltation, and *vice versa*, separated or not by an apparent "lucid interval." The depression may vary between a mere sluggishness or stupor to deep melancholia. The exaltation may appear in any degree from simple excitement to acute mania. The transition from one condition to the other may be sudden, frequently taking place at night, or it may be gradual. The length of the vicious circle varies or the duration of the succeeding mental conditions varies, but the type of each remains identical, or at least changes very slightly. The cycle may be completed in a day or two, or it may extend over several years (Folsom). Hammond recognizes two forms of the affection, one in which there is no intermission between the two stages; the other in which there are distinct periods of sanity between the accessions.

Gray's experience leads him to the opinion that "the lucid interval is not a true lucid interval. The patient is rather in a state of remission than of absolute inter

mission." Stearns says, "The patient passes into what may be termed neutral ground, between excitement and depression." This last named alienist has, in the opinion of this writer, given the clearest and most comprehensive description of the affection that he has yet read.

Blandford and Savage both are of the opinion that in any phase of recurrent insanity there may be a distinct period of convalescence between the attacks.

Dr. Daniel Clark, of Toronto, an alienist of considerable ability, says that he has never seen, in this affection, an intermission when the patient had normal mental health. In fact he does not believe there is ever an absolute recovery from insanity in any form,—that one is never the same after an attack of insanity as he was before.

Let it be remembered that to Falret and Baillarger, of France, each of whom published almost simultaneously, in 1853 or 1854, an article upon the subject, belongs alike the credit for first recognizing and describing *folie circulaire*, or *folie à double forme*, as a distinct genus of mental alienation.

The following illustrative cases embody the salient points of the disease:

CASE I.—Nancy S., a negress, 34 years old, has been an inmate of this institution since August, 1883. As is frequently the case with patients sent here, little is known of the family history. She was not married, had never had children; had had syphilis. Further than that nothing was known of her before she came under our care and treatment.

The period of depression is characterized by a torpid, sluggish condition of both mind and body. She is in a state of mental inertia. Differing from simple melancholia, there seems to be no mental pain. The cerebral faculties are almost entirely suspended. No illusions or delirium, delusions or hallucinations appear to harass her. She is quiet, her face almost expressionless; does not talk nor move about; is apathetic and indifferent—nothing excites or arouses her. If spoken to, she exhibits a silly expression of either surprise or absolute apathy. There is pronounced amnesia. All identity of persons, time or places is lost, the patient being unable to tell her own name or that of any one else, where she is, or whether it is night or day. In the most automatic way she does, when told, certain simple acts, such as going to meals or sewing for a few minutes.

She pays no attention to her personal appearance, neglects her toilet, is not tidy, etc. Her appetite is impaired, bowels costive, skin dry, pulse small and compressible, eye-lids droop, particularly the left, hearing dull. She sleeps well. This condition lasts about eight weeks.

From this state of stupid depression and inactivity the patient suddenly emerges into one entirely opposite, that of excitement and activity. The metamorphosis always takes place during the night. She is now in high spirits, skips and dances about the wards and yards, shows interest in her surroundings, answers questions more or less coherently. Her face becomes mobile and expressive, eyes bright. She is talkative, frequently going from one subject to another. She manifests erotic tendencies to a slight degree. Industrious and energetic, she is of considerable assistance in keeping the wards and rooms in order. She is now neat in person and dress, though she bedecks herself from head to foot with fancy articles of clothing, beads, ribbons, paper ornaments, and the like, presenting a most fantastical and grotesque appearance. She is quite generous in offering one her "pretty things," but begs for every bright-colored article of clothing she happens to see. A childish fondness for toys takes possession of her. She makes many rag dolls of various sizes and description. Indeed, she presents many eccentricities. Her physical condition improves, skin becomes moist, pulse full, hearing normal, bowels regular, appetite good. Her restlessness in the day frequently continues at night, disturbing her sleep. The duration of this period extends over about the same time that the stage of depression does. It takes, then, about four (4) months to complete the cycle. This regular alternation of depression and excitement has been going on ever since her admission to this institution in May, 1883.

The transition has always been immediate, the exhilaration following the depression, and *vice versa*—no period of intermission coming between the two morbid mental conditions.

CASE II.—William F., a negro, 36 years old, of fair education, steady, sober habits, was seized with gloomy depression a few weeks prior to his admission to this hospital, in September, 1886. This condition came on after a spell of fever. He was a stranger in the vicinity and no information scarcely could be gotten regarding his antecedents. When admitted he was in a state of



melancholic hypochondriasis; was the very picture of abject misery. Many imaginary ills troubled his peace of mind. He spoke of committing suicide, but evidently for the purpose of attracting attention and sympathy. On one occasion he said he intended to kill himself, but when the means to do so were placed at his command, he said he would do the deed at another time. The most trivial physical disturbances were exaggerated into very serious diseases. From this state of morbid depression he slowly emerged—grew brighter, more energetic, neater in personal appearance, etc. He was, during this period of slow transition or partial sanity, taken out on the farm where he proved to be a careful and industrious laborer. He escaped, and when brought back to the hospital a few weeks subsequently he was in a condition of great excitement and hilarity. His expression was animated, and he was, as it were, overflowing with super-abundance of spirit, very loquacious and incessantly moving. He bore an air of great importance and self-satisfaction; said he felt perfectly well and happy, but abused the officers for keeping him "confined unjustly in a lunatic asylum." It was his habit almost daily, if not interfered with, to deliver a long harangue to his fellow-patients, during which he would become very excited and noisy. He showed evidences of having a remarkable memory, particularly regarding names and dates. (Unusual memory is frequently observed in this type of sanity, says Stearns). He was sometimes disposed to be somewhat destructive to furniture, etc.; was neat in person, but would frequently dress rather "gorgeously," wearing feathers and the like in his hat, etc. He was not often noisy and sleepless at night, and then only for a short time. His physical health was good. This "mental intoxication," as it were, lasted nearly a year. After this long exacerbation of excitement there was a short remission and then depression again sat in which lasted about 15 months. At this time this patient is in the depressed stage or period of the third circle. So, thus the cycles have continuously repeated their weary rounds, and in all probability they will keep this up "until the final capitation in the battle of life has taken place."

CASE III.—H. J., a mulatto woman, was born about thirty-four years ago. She was fairly well educated, and at the age of twenty-two married an intelligent negro who has provided well for her. They have several children. Her mother was insane for a number of years.

From her husband I get the following facts: One night in the spring of 1892 she showed for the first time some signs of mental aberration, but soon became quiet and went to sleep. Later in the night she awoke, stole from her bed, laboring under the delusion that something dreadful would happen unless she go at once to a certain city, twenty miles distant, and secure the intervention of a noted politician. She tried to hitch her husband's horse, but, failing in that, she started to make the journey on foot, fixing her eye, she said, on some particular star which she thought would guide her. In the suburbs of the city in which she lived she attempted to jump on a passing freight train, believing it would take her to her desired destination. She fell, uninjured, however, in a ditch by the road. The train hands went to her assistance, and found her in a cataleptic state. She was taken to a hospital where every means, including electricity, commonly employed in such cases, were brought into requisition, but to no avail. She was in a profound hystero-cataleptic stupor. However, in about fourteen hours she suddenly aroused and gave her husband a correct detailed account of all that had happened. She became after this melancholy, insisted upon being left alone, refused to speak and took no interest in anything. After a few weeks of depression she became maniacal, jumped out of a window, a distance of twelve feet, and was quite excited for some weeks. She went for the second time into a state of deep depression. Again becoming morbidly excited, she went out on the street improperly clad, called upon numbers of people for the loan of money to take her away from home, saying she was cruelly treated there.

In January, 1893, she was admitted into this institution, and her general appearance indicated a thorough physical and mental depreciation. She was in the third month of gestation. She was pale and anæmic, with an icteric tinge of skin; kidneys and bowels were in a torpid condition; urine scanty and albuminous; pulse small and compressible; skin dry, and tongue furred. The characteristic depressing symptoms of *melancholia agitata* were readily observed. She could not be induced to leave her bed in the day for fear of bodily harm. At night her sleep was disturbed by horrid delusions and hallucinations of sight and hearing. Said she had committed "the unpardonable sin" and her soul was "eternally lost." She was the picture of despair, and sometimes would beg for some deadly drug or a weapon

wherewith to put an end to her "miserable life." It was with difficulty that she could be induced to eat, because, she said, she wanted to die. Her husband, she declared, was cruel to her and unfaithful to his marriage vow, and her children had been stolen from her by her husband's concubine. (All this, of course, was untrue.) There were occasional violent outbreaks during which she manifested homicidal impulses. Præcordial fear, the peculiar facies, insomnia and post-cervical ache were all present in a more or less intense degree.

After six or eight weeks had elapsed, by gradual transformation our patient became exuberant, garrulous, slightly incoherent, restless, and at times destructive. This excitement or mania lasted about a month, when she appeared to be in almost a normal condition, mentally and physically. She was taken home by her husband, for whom she then showed much affection. This was in May, 1893. A short time after reaching home she gave birth to a full term child, which, however, soon died.

This remission, or period of partial sanity, was of short duration, for our patient soon lapsed into a state of melancholia, which again in due time was succeeded by mania, and so on. Her husband wrote that the melancholia and mania—each period being about six weeks, it taking three months to complete the cycle—alternated with more or less regularity till she was again admitted to this hospital. The nature of the attacks was like those she had had previously.

On November 3, 1894, she was readmitted to this institution in about the same condition that she was in at the time of her first admission. She refused to talk, eat or move for the first few days. She gradually passed out of this state into that of maniacal excitement. She became noisy, filthy, destructive, profane and vulgar, with some erotic tendencies. Hypnotics were frequently necessary to induce sleep. At this writing she is much improved mentally, is perfectly calm, sleeps well, reads, talks coherently, etc. There has been a decided improvement in her physical condition. From the initial attack, in the spring of 1892, this woman has manifested an insane jealousy regarding her husband, whom she says she hates, and will not acknowledge as her husband.

#### CONCLUSIONS.

From observation and analysis of these cases, I conclude: First, that circular insanity is a rare but distinct

form of mental disease. Secondly, that to correctly diagnose a case it is necessary to have the patient under close observation for a considerable time, in order to note the peculiar and ever-recurring changes characteristic of the disease. Thirdly, that there is probably no period of absolute convalescence between the attacks. Fourthly, that the excitement may vary in intensity from a simple state of self-satisfied feeling to maniacal excitement, but the incoherence of speech and confusion of ideas are not so marked as in ordinary mania. Fifthly, that the depression may range from simple torpor to obstinate melancholia with suicidal tendencies. Sixthly, that there may or may not be delusions, hallucinations, etc. Seventhly, that there is nothing distinctive as a cause of the affection, for in one of my cases it was heredity, in another syphilis, and in the third probably fever. Eighthly, that though the symptoms in some instances may be improved temporarily, no system of treatment has yet proved to be of any permanent benefit in the disease.

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## FURTHER REPORT OF OPERATION FOR EPILEPSY SEVEN YEARS AFTER. COMPLETE RECOVERY.<sup>1</sup>

BY DR. J. MADISON TAYLOR

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IT may be interesting to see and examine a case of epilepsy operated upon with such absolute success. This boy, or rather young man, I reported five years ago.<sup>2</sup> It occurred in the service of Dr. Weir Mitchell, and the operation was performed by Dr. T. G. Morton in the Orthopædic Hospital. The early history in brief was this: W. B., a perfectly healthy boy, of wholesome antecedents, fell at the age of two years and eleven months a distance of fifteen or eighteen feet, fracturing his skull over the right parietal prominence; no convulsion at the time, and the wound promptly healed. Soon after there began occasional slight spasms, beginning with an aura, starting up the left forefinger and thumb, and limited to the arm. Later these increased, and a fuller description from the young man himself differs somewhat from the earlier notes, but is probably accurate because he is intelligent and quite positive on the subject. These smaller spasms the parents considered outgrown, and ended for a year or two. The first general convulsion occurred at the age of about ten years while in school, being rather excited over an unusual effort in arithmetic. While working very hard to accomplish the task, he noticed a greatly increased activity in the spasmodic opening and closing of the left forefinger and thumb, a phenomenon observed before, but held of small account. Presently the whole hand stiffened, the fingers clenched, the wrist flexed violently upon the arm, the arm flexed at the elbow, and was carried forcibly up under the axilla; the left side of the mouth was drawn down, then the left leg became some-

<sup>1</sup>Case exhibited at the Philadelphia Neurological Society, December 17, 1894.

<sup>2</sup>Ann. Gyn. and Paediatrics, August, 1890.

what like the arm, and finally he fell. Other attacks, usually superinduced by some mental or emotional excitement, occurred like this once or twice a month. The aura gave from a minute to a minute and a half warning, and the attacks could often be successfully controlled by a powerful effort on the patient's part or that of his parents to unlock the contracted parts persistently with the right hand, and to rub the palm vigorously. After the convulsions, during the period in which they were most severe, both the arm and leg were entirely paralyzed as to motion and sensation for an hour or two, then rapidly recovered. The boy was conscious throughout the seizure. He could hear and see plainly, yet was unable to move or speak, soon becoming vividly conscious of the absence of the arm and leg as the paralysis grew complete, and he would habitually ask to have the damaged arm placed in the sound hand so as to assure himself of its presence. When in the progress of the seizure the inevitable fall was about to occur the lad was always able to guide himself in a way so as to avoid receiving undue damages, and to especially protect the tender spot in the head by partly adjusting himself. If he were to lie on the right side the convulsions would continue, and it was necessary that he should be turned on his left for it to cease. Cold water or ice applied to the tender spot had the effect of limiting the convulsion.

Examination of the lesion, December 12, 1886 (this was before the crytometer had come into use), showed absence of bone over an irregular area (see diagram which I made at the time of the first examination) about two and one-half inches long vertically, starting two inches behind and in a horizontal line with the top of the external meatus of the right ear, and extending to within an inch of the vertex. The opening at its greatest width above the middle line was about one and three-quarter inches. On the posterior edge of the opening was a pointed mass of bone, elastic to the touch, pressure upon which produced a most sickening pain, and if continued induced a severe general convulsion. Also at that time the left hand and arm were of lessened development and vigor; not so the leg; knee-jerk excessive on both sides; station good; heart perfectly sound.

Examination of the eyes by Dr. George de Schweinitz showed the following:

Vision, each eye.—Amplitude of accommodation

diminished  $3^{\circ}$ . Reflex and associated movements of the iris normal. No muscular anomalies, except insufficiency of the internal recti of  $6^{\circ}$ .

Ophthalmoscope.—Right eye: Round nerve healthy color. Slight absorbing conus, outer side. Along the nasal artery small black spots, the questionable remains of hæmorrhage. Refraction II. Left eye normal, natural color; blotchy pigment conus at outer side; veins somewhat tortuous; macula normal. Refraction II. Central color perception good; fields of vision for form and color normal.

Diagnosis.—Hypermetropia and slight astigmatism, with insufficiency of internal recti. No appearance in fundus not explained by this error of refraction.

Operation March, 1887.—Dr. Morton, on lifting the scalp, found no spicules of bone projecting into the brain, but owing to the irregularities of the edges of the bony opening, certain pointed masses pressed severely upon the cortical substance. These acted as obvious causes for the convulsions, and they were carefully trimmed away, not disturbing the dura. No bone was replaced the scalp was carefully readjusted. The recovery was uninterrupted and rapid.

Description of W. B., December 17, 1894, as exhibited to the Philadelphia Neurological Society: Aged twenty-one and a half years, height five feet seven, weight one hundred and thirty-five; has enjoyed perfectly good health since the operation; is a powerful, muscular mechanic, earning an excellent livelihood as an artistic tile-setter. There has been no convulsion, except possibly a partial one soon after the operation, on which point is some doubt. The opening in the skull is still about as large as described in 1890. This is a depressed area, pulsating regularly, varying in depth in accordance with trifling disturbances of health. He very rarely has headaches, and only when touched or struck on this spot; then the pain is localized there. No considerable blow has been received, except one about two months ago. A companion playfully threw a soaked paper ball the size of an orange, striking him fairly on the tender place. Falling instantly to the floor, he suffered for some hours thereafter intense pain. The left arm is about three-eighths of an inch less in length than the right; but the chief difference resides in the hand, the left being fully a half inch shorter than the right, and the whole member proportionately small. The legs seem

precisely similar: the buttock lines are symmetrical; the left foot is smaller than the right, but no more, perhaps, than is natural.

The points of special interest here are: First, the entire relief to the convulsions. Second, perfect recovery of functional activity of the limbs. Third, entire absence of spasticity or other damage to the motor tracts except only the lessened development of the one arm and hand which is confined to size and not modifying strength to any considerable extent. Fourth, perfect co-ordination, normal growth and development of intelligence.

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**The Therapeutic Utility of Trional.**—Dr. B. Rohmer. Inaugural Dissertation presented to the University of Strassburg, 1894. The remedy was employed in twenty-nine cases in which sleep, was disturbed by restlessness, bodily pains or irritating cough. In all but one case trional was found to possess hypnotic properties, and in twelve of these the effect was always the same, although the period of administration extended over several weeks. In seven cases the effect was weakened or lost in the course of time, necessitating an increase of dose. In some cases the apparent cause of this was the presence of bodily pains which exerted an inhibitory influence upon the action of Trional. Thus, in a case where sleep of ten hours' duration resulted from the 15 grains given the first evening, the effect of the same dose given on the following day was completely counteracted by the presence of toothache. That the pain must, however, attain a more or less high degree of intensity in order to neutralize the action of the remedy is demonstrated by a case of *tuberculous dorsalis* in which sleep could not be induced by morphine, but followed administration of one-half drachm trional. The author's experience further demonstrates that the action of a single dose may extend over several nights, although he was unable to confirm the observations of others that the effect is developed on the average in the course of half an hour. Even in the case where its action was pronounced, sleep frequently failed to appear until the end of two hours. The duration of the effect varied from two and one-half to ten hours. Sleep was deep and quiet and the patients rarely experienced any disturbances on waking. Judging from its behavior in two cases of cardiac disease, trional seems to be devoid of tonic influence upon the circulatory system. After prolonged administration no disturbances resulted when it was discontinued. Rohmer's observations conform, therefore, in the main to the experience of other observers. He regards the remedy as especially indicated in psychiatric practice, and recommends its use in simple insomnia and even where this condition is due to pain. The doses varied from a half to two scruples given in wafers. J. C.



## NOTE ON A CASE OF TETANUS.

By PHILIP MEIROWITZ, M.D.,

Assistant in Nervous Department, Post-Graduate Medical School.

M. G., schoolboy, aged 12 years; both parents living and healthy; the family history is negative in respect of nervous and mental diseases. The patient himself has been exceptionally healthy, not having experienced any of the infectious diseases of childhood. I was called to see the boy on the third of November. His mother informed me that three weeks before, the boy had received a beating at the hands of a neighbor, who stood the boy on his head and struck his back. No bruise or break of the skin was visible, and the boy soon recovered from the effects of the thrashing. A week later the patient ran a splinter into his hand, which was extracted by his mother, who treated the wound with the usual household remedies. The wound healed in a few days. During the week following the boy enjoyed excellent health. On October 27, a week after receiving the splinter in the hand, the boy complained of some languor, some stiffness in the arms and in the maxillary joints. He could not open the jaws quite as wide as he could the day before, and requested that his victuals be cut into small pieces for him; he was still able to romp about, and did not cease from his play until 9 p.m., when he retired to rest with the symptoms above mentioned, which were not sufficiently marked to cause the parents alarm. The following morning he was unable to leave his bed. He was discovered with stiffened and outstretched extremities and locked jaw. He had had no fever or convulsions, and had not complained of headache; nor had there been any loss of consciousness. Physicians were summoned, and treated the boy for five days without improving his status. I was called in on the sixth day and found the boy in the following condition: *Risus sardonicus* was well marked; the head, feet, legs and thighs

were rigid and extremely extended; the latter were adducted; the abdominal muscles were contracted into a board-like condition; the right arm was extended, but to a less degree than the lower extremities; the left arm was semi-flexed; opisthotonos was well developed. Passive motion gave rise to muscular spasm. The reflexes were exaggerated, but there was no ankle clonus, owing to the extreme contraction of the muscles of the calf. The respiration was in the main regular, but at times it exhibited the Cheyne-Stokes type. The pupils were of medium size and reacted to light. Owing to the spasm of the maxillary muscles, it was difficult to introduce food or medicines into the mouth. At short intervals the tonic spasm in the already contracted muscles would become increased, the opisthotonos more marked, and the risus sardonicus more accentuated. The increased tonicidity would last a few minutes, then relax. At all times consciousness was completely retained. The pain accompanying the spasm was moderate, but became more severe, with the recurrent increased tonicidity, causing the patient to implore assistance. The following day, November 6, the patient died in a comatose condition. The pulse had become progressively weaker, the extremities cold, the pupils dilated, and the opisthotonos extreme. After the heart had ceased to beat, deep inspiration occurred from time to time for a period of five minutes.

When first called to the case I attempted to secure relaxation of the contracted muscles by chloroform inhalations, but no sooner had a few whiffs of the anæsthetic been taken than alarming Cheyne-Stokes respiration developed, and I was compelled to desist. I then resorted to curare, hyoscyamine hydrochlorate, and morphine, subcutaneously with but slight effect. Then thirty grains of chloral, together with half an ounce of whisky and eight ounces of milk, were given per rectum, with the result that sleep and some relaxation of the muscles were produced; but the reflex irritability of the nervous system still persisting, another dose of thirty grains of chloral was given six hours after the first. It was in the continued sleep, produced by the second dose of chloral, that the patient succumbed.

So far as I could learn, no attempt had been made to use chloroform in the early stages of the disease; nor had feeding per rectum been instituted. It seems to me that the two main indications in the treatment of tetanus are, to reduce or abolish the enormous irritability

of the nervous centres, and to maintain the strength of the patient. To accomplish the former, one is justified in pushing the narcotics and anodynes employed, to the extent that the patient be kept continually in a semi-comatose condition. The strength of the patient may be easily maintained by injecting sufficient quantities of nutriment into the rectum. As no tetanus anti-toxine could be obtained this remedy was not used.

As regards the etiology of the disease in this particular case, it would be interesting to know whether the wound inflicted on the hand by the splinter was at all responsible, whether the beating which the boy received had predisposed his nervous system to the infectious agent, or whether the tetanus bacillus had gained entrance to the blood through some channel other than the break in the skin.

**Chorea as a Fatal Disease.**—Barber (*Brooklyn Med. Jour.*, February, 1895). This disease is not considered by the mass of practitioners as one needing especial attention, and few authorities consider it of sufficient gravity to even suggest that a case might end fatally. The British Medical Association computed the deaths at about two per cent. The author's first fatal case was that of a six-year-old boy. He had been ill for two weeks, and twitchings were first noticed about the mouth, the spasmodic movements gradually increasing, until the whole body became more or less involved. A physician had advised that he be urged to move about, and in consequence he showed scars and bruises due to falls. The spasmodic movements became excessive, he only was able with great effort to converse and could not masticate any kind of food. Only liquid nourishment in very small quantities was taken, as the interval between the spasmodic seizures was brief. After a few days of uninterrupted distress he succumbed to the disease. Autopsy revealed nothing characteristic in any of the viscera or nervous structures. There was no history of rheumatism, and the patient had been well until attacked by this disease. He died from asthenia. A second case was that of a man eighteen years old. His first attack came on two days after being thrown from a wagon. After a few weeks' hospital treatment, he recovered. The second seizure came on just after being admitted to bail, the patient having been arrested and locked up for lounging. Involuntary movements appeared and grew very severe, so that it required the attention of three men to hold him in bed. The mind was clear, and during sleep the movements ceased. He died from the same cause as the first case after five days. No post-mortem examination was allowed. FREEMAN.

## A PROBLEM IN NEUROLOGY.—PECULIAR IRIS-REACTION WITH POST-NEURITIC OPTIC ATROPHY.<sup>1</sup>

BY DR. GEORGE M. GOULD,

Philadelphia.

OMITTING unimportant details, the principal facts necessary to understanding the proposed problem are these: My patient is a girl of twelve years of age who had been afflicted with obscure spinal and cerebral disease prior to being sent to me for diagnosis and treatment of the ocular conditions. I saw the child for the first time about one year and one-half ago, at which time there was complete blindness, stabile mydriasis, and most intense neuro-retinitis, both papillæ being enormously swollen, the retinal vessels congested, tortuous, and often hidden by the inflamed retina and by multiple scattered hæmorrhages. Both eyes were in the same condition.

Six months later, at the second visit, the neuro-retinitis had quieted down, and typical optic atrophy was pronounced, though vestiges of inflammation existed. Not the faintest perception of light was present.

At the third visit, about one year from the date of the first, there was absolute optic atrophy of the typical white variety, stabile mydriasis, so far as relates to the stimulus of artificial light, and complete blindness. The most brilliant and concentrated light thrown suddenly or continuously through the pupil failed utterly to elicit iris-reaction or perception-response. But, by seating the child before an open window, the street in front being illumined by sun light or diffuse daylight, within half a minute or more the pupils were found to be of normal size. The parents had noticed that when the child had played out of doors the pupils were of the size usual in other people. The motion of the contraction was too

<sup>1</sup> From the Transactions of the Eighth International Ophthalmologic Congress.

slow to observe, that is, one could not positively say that the myotic movement was taking place by watching the pupil attentively. In the same way, the widening, when the patient's face was turned away from the window and directed toward a moderately lighted room, was perhaps more rapid, but still too slow to detect its progression.

The problem, a double one, is, of course, this: (*a*) How can the optic nerve be the afferent intermediate of pupillary response when following a peripheral atrophying neuritis that produces total blindness? And (*b*) Why does the pupil react to the stimulus of continuous diffuse daylight, and not to that of the most brilliant artificial light?

A number of alternative queries arise. For example, is it possible: (1) That there is a localized molecular action of daylight upon the muscular iris-fibres distinct from central neural connection and control? (2) That the stimulus, generalized, strong and continuous, of the daylight is powerful enough to carry some nervous impulse through the atrophied nerve fibres, and so far as the pupillary centres, but that this impulse is too weak to reach the visual centres? (3) Had there been originally a synchronous atrophying lesion of the optic centres or of the conducting paths beyond the pupillary centres (which would explain the blindness), but that still left a few fibres intact between the retina and the corpora quadrigemina? (4) Is there some hitherto unproved neural connection between the iris *per se* and the quadrigeminal bodies? (5) Is the neural intermediate by means of the fifth nerve? Or (6), and finally, is the visual centre stimulated *via* fibres direct from the retina, and not by fibres from the pupillary centre; in other words, are there distinct fibres that proceed to the pupillary centre and end there, the neural impulse not proceeding hence to the visual centres, whilst other distinct fibres proceed directly from the retina to the occipital lobe without calling at the pupillary centres? The fibres of the latter class in my case being all atrophic, while some few of the first class escaped unharmed, or so injured as to respond only to the continuous daylight stimulus.

In this connection I may allude to a case I described in the "Philadelphia Hospital Reports," Vol. II., 1893. A microcephalic child, with congenital cataract, according to my own repeated observations, as well as those of others, had definite light-perception when artificial

light was concentrated on the pupil, and as certainly was there a slight iris-reaction, and yet the post-mortem examination of the optic chiasm by a competent histologist unmistakably proved to his mind a complete atrophy of all the nerve fibres.

Since writing the foregoing I learn that Dr. Myles Standish, of Boston, has had a case, as yet unreported, similar to mine.

*The Bromides Combined with Adonis Vernalis in the Treatment of Epilepsy.*—Bechterew (*Neurolog. Centrbl.* No. 23, 1894).

"In an epileptic attack there are vaso-motor changes in the brain, characterized by an active hyperemia within the cranial cavity. It is on this supposition that the activity of the bromides can be explained. Therefore, it appears to me that it is serviceable to combine the bromides with such a substance that would at once increase the blood-pressure and at the same time narrow the lumen of the blood vessels. Such a substance is represented by *adonis vernalis*." Possessed of this idea, the author has used *adonis vernalis* for a number of years, and believes that it has virtues sufficient to warrant calling the attention of the profession to the matter. The mixture which he uses is prepared by making an infusion of *adonis vernalis* from 2.00-3.75 to 180.0, straining, and adding from 7.50-11.25 of bromide. Of this mixture from four to six, and even eight teaspoonfuls are given daily. If a sedative is indicated, from 12 to 18 ctgrm. of codeine are added to this mixture—the 180 grammes. After considerable experience with this plan, the author says he feels called upon to say that not infrequently the attacks are checked, while in other cases the number and severity are very much lessened. The salutary effects of the treatment are so apparent to the author that he says: "In the many years of my practice I have yet to see a case of epilepsy in which the systematic use of the above mixture did not show its favorable influence." If it were not for the cumulative effect of the infusion of digitalis the combination of it with bromide would be quite as serviceable.

J. C.

## A CASE OF WAITERS' PARALYSIS.

BY EDWARD C. RUNGE, M. D.,

St. Louis, Mo.

J. C., æt. 30, single, restaurant waiter, applied for treatment at the Clinic of Diseases of the Nervous System of the St. Louis Medical College on October 13, 1894. The inquiry into the patient's family history brought out the fact of his father's death in an accident, of his mother's death in a way unknown to him, and of the existence of a case of tuberculosis on the maternal side of the family. The patient has always been a moderate beer drinker. His previous health has apparently been good. He contracted syphilis seven years ago, and received specific treatment for seven or eight weeks; he claims to have not experienced any luetic manifestations since.

His general condition at the time of his first visit was as good as could be desired; his appetite fair, bowels regular, micturition and defecation normal. He did not complain of any sensory disturbances or headache. About twelve months ago, while engaged in the pursuit of his occupation as a waiter, his wrist gave out suddenly. He was in the habit of carrying a large number of dishes piled up along the whole extent of his left arm from hand to shoulder—a fashion to be readily observed in any of the cheaper eating houses. The arm is extended and supinated, the hand strongly adducted, with the palm upwards. The patient states that on that occasion, without the slightest warning, his arm assumed suddenly the position of pronation, the hand, of course, turning palm downward, and his wrist dropped, thus precipitating to the ground the dishes carried at the time. The description pointed clearly to a paresis of the supinator and extensor muscles. The patient asserts that the wrist never gave him any trouble unless he attempted to carry the dishes in the habitual manner, when it invariably gave way.

The physical examination resulted negatively, except for a sluggishness of the right pupillary light reflex. No motor or sensory disturbances could be made out; co-ordination, muscular power, tendon and skin reflexes seemed unimpaired; the electrical tests failed to show any reaction of degeneration. No manifest signs or symptoms of a still active lues were discernible.

Up to November 15, 1894, he has been receiving local galvanic treatment thrice a week. No internal medication was attempted, except for a very short course of strychnia at the beginning. On the last mentioned date the patient stated that he had returned to his old occupation, and that he was only reminded of the wrist trouble by a sensation of weakness whenever he worked steadily for an hour or more.

This case evidently belongs to the class of so-called occupation neuroses. While reports of similar disturbances in writers, piano players, telegraph operators, cigar makers, tailors and dairymaids, have been made I am not aware that a case of—what I choose to call—waiter's paralysis has been placed on record before this.

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***Abscess of the Left Occipital Lobe, Causing Object-blindness, Word-blindness, Etc.***—Campbell (*Liverpool Medico-Chirurgical Journal*, January, 1895).

A 43-year-old temperate man had been deaf in the left ear for thirty years, and for the last ten years this ear had discharged. On otoscopic examination, a perforated membrana tympani and granulations were detected, and otitis diagnosed. This was probably the origin of the cerebral abscess which caused his death. The symptoms developed after several attacks of dizziness and consisted of; 1. Object or mind-blindness. 2. Word-blindness and paralexia. 3. Paragraphia. 4. Hemiplegia. 5. Seizures followed by temporary absolute aphasia and paresis of the right arm. 6. Absence of sensory anomalies and permanent paralysis. 7. Signs pointing to an involvement of the cerebellum, such as rotatory nystagmus in all directions, ataxia and weakness of the trunk muscles. 8. Delusions of persecution, visual hallucinations. 9. Double optic neuritis with early hemorrhages.

The post-mortem revealed: 1. An abscess destroying the greater part of the left occipital lobe. 2. Softening of the upper surface of the cerebellum. 3. Degeneration in the postero-external columns of the spinal cord.

J. C.



## Asylum Notes.

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By R. M. PHELPS, M.D.

Rochester, Minn.

*Sketch of the Rochester State Hospital for the Insane.*—So many distorted and queer accounts of insane hospital affairs are common that a plain account of the methods and work in one may be corrective and interesting. It is in this spirit that we offer the following very brief detail concerning work in the Rochester State Hospital:

This hospital, built upon the common radiating ward plan, accommodates at present 1,150 patients in buildings nominally holding less than 1,000. This discrepancy is due, as is commonly the case, to the length of time necessary to make legislative appropriations into actual buildings. The hospital has grown up since 1880, and, therefore, has been quite steadily building. Besides the central building there are two annex buildings, one for men (290), one for women (209), built for economy's sake upon the plan of "one day flat, one dining room and kitchen flat, and two flats for sleeping." About 700 acres of land are owned or rented, and the hospital is situated in a very pleasant valley about one and one-half miles from the centre of a city of 5,000 people. The usual supply of barns, farm buildings, green-house, ice-house, carpenter shop, store house, steward's general store, slaughter house, etc., are provided, and furnish us with about the ordinary outfit for insane hospital work.

The medical authority is vested in a superintendent, an assistant superintendent, and three other assistants. The administrative work is vested in the superintendent, and the financial work, under direction of the superintendent, in an accounting officer, or steward. The hospital work (the central work, to which others are merely supply) is that, of course, which we more especially wish to describe. Hospital work here, as elsewhere, is partly medical, and partly clerical and administrative. The rounds of each ward are made twice each day by the assistant in charge of the section, from two to four

hours of the day being so spent. The assistant superintendent also makes the complete round daily, and the superintendent also, as his time will permit.

The patient, upon being admitted into the hospital, after being received upon the ward, given a bath, and due notes made of any physical peculiarities, is put through an entrance examination, which, altogether takes from three-quarters of an hour to two hours. A synoptical sheet is provided to secure the assistant against forgetting any organ of the physical system or any element in his mental condition. Such elements as examination of urine are, of course, not made exactly at the same time, but within a short time afterwards. The line of treatment is quite definitely indicated by this review. Patients are quite commonly put in bed for a few days, even if the indication for it is not especially pronounced, as they are thereby watched more closely, and more powerful impression given that they are under hospital treatment.

For the succeeding accounts, and for all daily observations, we are greatly helped by our trained nurses and our school for the same.

Although the school was not established for such reports, yet we have made much of it. Early being impressed with the idea that the expansion of our work must depend upon the nurses, and that without them progress must, perforce, be limited, we have made much of the training of nurses, and with very full response. For example, they take something like one hundred temperatures a day, and as each temperature takes from five to ten minutes, this would be impossible for assistants to find time for. But this is only one element. They give enemata, hypodermics, catheterize, dress ulcers, apply poultices and fomentations, give eye and ear applications, care for surgical cases, do all the preparations for surgical operations, attend to all anti-septic and disinfecting measures, do all of the mechanical work in giving electricity, while a specially selected class of them have studied and give massage. They also procure and examine specimens of urine, and present results to us for confirmation. Two of the leaders, one male and one female, have taken special lessons in cooking, to preparing delicacies for those physically and mentally sick, and in order to instruct the others.

Moreover, three of the more ambitious ones are taking up the microscopic examination of the sediment of

urine and the preparation and examination of tubercular sputa. They prepare and present the specimen to the physician for examination. We aim at nothing less than the full ideal of training school work elsewhere, and let our best nurses go out on cases of private nursing, when we can spare them, and have always some thus employed.

It will be readily seen that an amount of work of medical observation and records is made which five times the number of physicians could not have accomplished with the former service—such service as could not do the work designated above. But, more than this, they record all observation and work, and for the past three years the division of labor has been applied here also. Nurses are taught to describe accurately ordinary behavior, delusions, incoherence, etc., as well as all physical symptoms and peculiarities, making thus a good record of all but the most purely medical ideas. Each patient has, accompanying him to the ward, a fourteen-day record blank, with synoptical headings, suggesting a canvass of all peculiarities, mental and physical. After the patient has been here fourteen days an ordinary blank replaces this, upon which a canvass of the patient's condition is recorded at least once a month, and notes made of all intervening happenings of importance. For those having especial physical disease an ordinary nurse's ruled blank is given also. Ordinary blanks are handed in every three months. This brings into the office about 5,500 sheets of records a year, which immense literature, after being reduced to about one-third by cutting out repetitions and needless matter, is copied into the case book by a clinical clerk, and is a work more than sufficient to fill one man's time. Physicians' notes can be added to those of the nurse, or handed in separately. An average of about one patient a day is received.

Patients are classified in so crowded a place upon many principles. Recognizing that a large proportion of them are chronic before admission, not all go upon "admission" wards. For the sake of room, new admissions and suicidal patients are in the same ward, as both need especial care and observation. Senile dementes, the old and weak, are upon the ground floor, so as to easily get out with less danger of injury. The especially untidy have a ward for themselves, the epileptics a ward, the violent and restless have two wards, while the an-

nexes hold a large number of quiet, chronic manias and dementias.

Upon each side of the house, male and female, is an infirmary ward, which we think compares favorably at present with any in the country. A large and well supplied operating room is on each of these wards. A Veters switchboard with batteries in basement is in each of these wards. A urinalysis outfit is in room adjoining the bath-room. A large gas stove in kitchen of each ward supplies chances for special cooking for the sick and those needing nourishing diet. A Turkish bath outfit is lately placed upon the male ward. There is a head nurse for each of these infirmary wards, as well as one for each side of the house. An average of about forty to fifty are daily in bed for sickness, or in order to conserve strength. Suitable acute cases of insanity as well as those bodily sick are treated upon this sick ward.

Employment is considerable. Some 500 people help in the laundry, kitchen, farm, bakery, garden, ward work and other industries, promoting greatly the economy of their care. Many others are occupied in more light and trifling ways upon the ward, still others have parole of the grounds, but do not wish to work. This accounts for about fifty per cent. Of the other fifty per cent., at a guess, one-half could be induced by close supervision and much overseeing, to do work, which would be valuable chiefly to the patients themselves.

Of the medical work not much of value can be said in small space. The routine work of the day beside that above mentioned, includes seeing and satisfying the daily visitors, keeping track of the daily heating, ventilation, and above all, the supervising the life of the patients and the conduct of the nurses. Then, there are occasional autopsies, also surgical operations, urinalyses, electrical and microscopical work. There is also the considerable work of the training school lectures, and the conduct of the school. To this if a physician add any original, collating or critical "working over" of his clinical observations and keeps up with current literature, he does well, indeed.

## Periscope.

### PHYSIOLOGICAL.

**A New Method of Dividing the Surface of the Cortex Cerebri.**—Flechsig. (*Neurolog. Centrbl.*, October 1, 1894.) In this preliminary communication Flechsig bases his remarks on the study of preparations made in his laboratory by Mdler. The convolutions of the human brain may be divided into two great divisions. The one includes those areas which receive or give origin to the sensory or motor fibres (optic radiations, pyramidal tracts, fillet, etc.), besides a few association fibres. The second division has no direct connection whatever with the corona radiata, and contains only association fibres, the corpus collasum and commissural fibres especially. The first area, Flechsig refers to as "sensory centres." They include the area for vision around the calcarine fissure, the area for hearing in the posterior portion of the first temporal convolution, the area for smell in the gyrus hippocampus, and the posterior part of the inferior surface of the frontal lobe, and lastly, the great central motor region about the fissure of Rolando, including the posterior portions of the frontal convolutions, or, in other words, the large area which embraces the posterior portion of the frontal convolutions and the central convolutions, and from which go out fibres to the pyramidal tracts, fibres to the great ganglia of the pons—"the frontal cortex-cerebri-pons pathway"—the bundle to the red nucleus of the tegmentum caudicis, to the upper cerebellar peduncle, the nuclei of the posterior columns, and probably also to the vestibular and trigeminus nuclei.

The second great class of areas, the "association centres," occupy those portions of the brain that have not been allotted specific function by the localizationists. On account of the different period when different nerve fibres become medullated, the extent of these areas is best studied in the brain of a three months old child. At this age, almost the entire corona radiata is medullated, and these streams of medullated fibres radiate out to distribute themselves in the sensory areas described under the first heading. Scarcely one-third of the cortex is thus supplied with medullated nerve fibres, and the large association areas which make up more than two-thirds of the brain surface are entirely destitute of medullated fibres, or contain only a few scattering fibres, which reach them, for the most part, through the sensory centres. They comprise four great tracts.

They are the anterior portion of the frontal lobe, the island of Reil, a large part of the parieto-occipital lobes, including the precuneus and posterior portion of the parietal lobe. It is probable that at this early stage of development each sensory centre possessed its own sensory mechanism distinct from every other. The association system which unites these areas one with another, or with neighboring sensory centres, are very much more numerous than the association system which unites sensory centres directly with sensory centres.

The greatest difference between the brain of man and that of other animals is found in the enormous development of the association centres. Their development influences in fact the form of the skull and the type of the brain. The speech centre in all seems to lie in the border of the association and sensory centres.

J. C.

**Cerebral Optical Centres.**—Dr. S. E. Henschen. (*Extrait de la Revue Générale D'Ophthalmologie*, No. 8, 1894.) Regarding localization and physiological functions of the cortical visual centre, physiologists differ. Munk limits it to the occipital lobe; Luciani and others have extended it to the angular gyrus and even farther; Ferrier and others localize it chiefly in the angular gyrus.

VALUE OF ANATOMICO-PATHOLOGICAL METHOD IN LOCALIZING THIS CENTRE.

In recent years different authors have tried to base their observations on the method of secondary degenerations in animals and man. H. cites an example: Male; blind for fifty years; both eyeballs destroyed, and there was found complete atrophy of the optic tract, considerable atrophy of the external geniculate body, pulvinar, anterior corpora quadrigemina, radiation of Gratiolet, and some atrophy extending to occipital, parietal and temporal lobes. He asks should we then consider that all the fibres are visual, conducting light impressions. We know that destruction of fibres which go to pulvinar, anterior corpora quadrigemina, temporal and parietal lobes does not cause blindness.

Degeneration in the opposite direction in the internal capsule from partial destruction of the occipital lobe is not for that reason to be considered visual fibre degeneration. Conclusions based alone on degeneration method are not reliable. Henschen believes that only the anatomico-clinical method can guide in localizing the optical centre. In trying to settle this question only positive cases of hemianopsia have been taken into account. In analyzing negative cases together with the positive, H. believes the optical centre to be limited to the calcarine fissure, but impossible to say which part. The occipital visual tract is located in the inferior part of the optical radiation. Hemianopsia, he says, arises only from a lesion of this fasciculus or of the calcarine fissure, and he has not found one case among those well reported militating *vs.* this theory.

Some have localized the centre in the parietal lobe, but there are many negative cases reported of lesions of this lobe without hemianopsia. The author gives a number of diagrams of regular cases, and calls attention to the fact that the cortical lesion of the parietal lobe causes hemianopsia only when it touches the visual fasciculus in the inferior part of the optic radiation.

A large number of anatomico-clinical facts show that the visual centre is limited to the occipital lobe. H. publishes diagrams of cases where lesions of the median surface have not produced blindness, but adds, cases few and lesions sometimes small.

An analysis of all the cases the author is able to collect convinces him that hemianopsia results only when the lesion involves the calcarine cortex or the optical fasciculus which unites the geniculate body to this part of the occipital lobe.

The exact limitations of the optical centre cannot be as yet definitely settled, but the author has observed one case when the lesion was limited to the cortex of the calcarine fissure and the hippocampus in a very precise manner, and the hemianopsia complete, thrombotic softening limited to the cortex hidden in the depth of the fissure. Microscopical examination showed secondary degeneration of the optic radiation. This case appears to be the only one where a lesion was limited to the cortex of the calcarine fissure.

Can we determine where the macula lutea and peripheral portions of the visual fields are projected? H. cites a case where the white substance of right hemisphere was almost totally destroyed, together with two and a half centimetres of the occipital part of the calcarine fissure of the left. Patient could read tolerably fine characters up to time of death. We read by macula lutea, hence this would seem to be projected to anterior part of the calcarine fissure.

II. also attempts to localize the superior and inferior part of the visual field. M. Monakow says there is no projection from the eye into the cerebral cortex; a new centre can form of itself if the peripheral cortical centre is destroyed; the centre then is mobile. In support of this theory analogies exist in the motor and sensory centres. H. meets this theory by asserting that hemianopsia caused by a cortical lesion in the calcarine fissure is not transitory, and claims this is supported by his own case and those of Hun, Déjerine and Wildbrand. H. does not think Monakow's case, which he comments on in detail, upsets in any way his conclusions. ELLIOTT.

**Experimental Researches on the Fatigue of the Human Muscles Caused by the Action of the "Nerve Poisons."**—C. Rossi. (*Rivista Sperimentale di Freniatria*, 1894, fasc. iii., iv., page 442.) The experiments were made upon the author himself and his laboratory servant. The muscles experimented upon were the flexor digiti medii of both hands. The researches were made in the following manner: A weight, which was uniform for all experiments, was raised (and lowered) every two seconds until the muscle was so tired that the weight could not be raised any more. Every elevation was marked on the "ergograph" of Mosso. Ten minutes after the starting of the first series of muscular contractions another series was started; ten minutes later a third, etc. The experiments were thus conducted during one hour, making seven series for each hand.

The following day repetition of the experiments in the same manner under the influence of the drug, which was taken by mouth or by hypodermatic injection directly before the beginning of the experimenting. In some cases, where the author desired to study the later effects of the drug, the sessions were prolonged to two hours or more.

As criterions served:

1. The mechanical labor performed (sum of single elevations and weight) (a) in each single series, (b) during a whole session.

2. The number of muscular contractions in one single series. The results attained were:

- I. Alcohol, absinth, caffeine, ether and strychnia increase the resistance to fatigue.

- a. Alcohol: Given in a considerable dose (80 grammes of rum), it first increases the resistance to fatigue, but soon (within half an hour) diminishes it. In small doses (25 grammes of rum) it increases the resistance to fatigue, which increase remains stationary as long as one is under the influence of the drug.

- b. Absinth: First highly increases the resistance to fatigue, an effect which lasts only from twenty to thirty minutes, and which gives place to a rapid diminishment. The absinth acts as a temporary excitant.

- c. Caffeine: It increases both the number of contractions in a single series, and the quantity of the total mechanical labor performed. This effect remains stationary as long as the drug acts; it is not followed by a diminishment of the resistance to fatigue. Caffeine must, therefore, be considered as a tonic for the neuro-muscular system.

Camphor: It displays a variety of action; in one person it increases the resistance to fatigue; in others it diminishes it. Its effect will always depend with the dose and the individual.

Strychnia: Increases the resistance to fatigue, which manifests itself not only by the increase of the later mechanical labor, but also by the increased number of single contractions.

- II. Atropine and hyoscyamine do not influence the resistance to fatigue in any manner.

- III. Potash, chloral hydrate, dulcisine, morphine and opium have a depressing effect, diminishing the resistance to fatigue.

a. Bromide of potash: The depressing effect is of short duration, the neuro-muscular activity very soon revives to return to the normal.

b. Chloral hydrate: The depressing effect is stronger and lasts much longer than that of bromide of potash. Both the later mechanical labor and the number of muscular contractions are diminished.

c. Duboisine: Acts similarly to chloral hydrate. The diminution of the mechanical labor is very intense.

d. Morphine and opium: The resistance to fatigue is diminished in a high degree. This diminishment is most pronounced at the beginning of the experiment series.

ONUF.

#### ANATOMICAL.

**A Finding of Anthropological Significance in the Negro Brain.**—Waldeyer (*Sitzungs-Berichte der könig. Preuss. ak. d. Wissensch. zu Berlin*, 1894, *Neurolog. Centrbl.*, Feb 15, 1895).

The author, after careful study of the Negro brain, comes to the following conclusions: The Sylvian fissure is characterized by its remarkable shortness. The steep ascent of the posterior end of this fissure which has been described by some authors, was not found constantly. The *pars opercularis* appeared frequently to be small and to sink in very deeply. In one specimen the *pars operculum* was very small. The central fissure the author found to correspond in almost all instances to the ordinary conditions. The central convolutions showed a tendency to excessive fissuration. Very significant were the findings in the sulcus parieto-occipitalis, which in one-half of the cases extended far out on the lateral surface of the hemispheres. The cuneus, lobus paracentralis and sulcus fornicatus showed no departure from that common to the Caucasian brain. The precuneus was found to be large in every instance. The average weight of the Negro brain was found by Topinard to be 1234 grammes. Waldeyer, in his examinations, found it to be 1148 grammes.

In conclusion, the author remarks that only from an examination of a large number of specimens can any trustworthy and reliable additions be made to the anthropological problem which his remarks have opened up.

J. C.

**Nomenclature in the Anatomy of the Nerve Cells.**—Nissl (*Neurologisches Centralblatt*, 1895, Nos. 2 and 3).

Based upon the reaction obtainable by his method of staining, Nissl proposes to speak of a "stained" (that is, visibly formed) and an "unstained" substance of the cell body of the nerve cell. For the stained substance a distinction of "intensely stained" and "slightly stained" parts ought to be made. The variety of forms under which the stained substance presents itself necessitates the introduction of names to characterize the constituents of the cells and their arrangement. N. proposes, therefore, to speak of "Körnchen," "Körnchen" groups "rows," "threads," and of "corpuscles." The corpuscles are mostly intensely stained and characterized by their large size, while the other formations mentioned are much more delicate, and stain less intensely. To characterize the various forms of the corpuscles the following expressions are proposed:

1. The "nucleus-caps" (Kernkappen). These are conical of regular or irregular shape, and with an excavated basis, adapting itself to the outline of the nucleus, and upon which the nucleus-cap sits.

2. The "ramification conical" (Verzweigungskegel). These are found where a process ramifies, and they fill out the angle between the ramifications.

3. The "spindles." They are thin and long, attenuating themselves in one or both directions, sometimes ending with a thread-like formation.

<sup>1</sup> Literally granules, but the author wishes to avoid the name "granula."



In all of these forms the so-called "corpuscle-vacuoles" (Körperchen-vacuolen) are frequently seen. They appear as small, light spots, with sharp outlines in the corpuscles.

The classification of the nerve cells is further based upon the development of the cell body. Many cases have a well marked cell body, inclosing the nucleus completely; in others the nucleus forms the principal part of the cell, the cell body is often reduced to one or two process-like attachments to the nucleus, while the rest of the latter is not surrounded by cell protoplasm (for instance, cells of the substantia gelatinosa Rolando). For the cells of this type, the nuclei of which do not exceed the neuroglia nuclei in size, N. proposes to retain the name "granuli" (Körner).

Those cells of the same type whose nuclei are larger than the neuroglia he proposes to call "nucleus cells" (Kernzellen). The various structure of the nuclei would further allow a distinction of the a, b, c, d, etc., types of the granuli and nucleus cells.

To Greek-lovers N. offers the names "karyochrome cells" instead of "nucleus cells," and "cyto-chrome cells" instead of "granuli." The cells with well-marked cell body would then be characterized by the expression "somato chrome cells."

In all these cell types the various "states of staining" necessitates a further distinction of

(a) Pyknomorphe Cells.—The stained part of the substance of the cell-body is densely arranged, giving the cell-body a dark appearance. In the nucleus the stained part (Kerngerüste) is usually also more strongly developed.

(b) Apyknomorphe Cells.—The stained part of the cell-body is loosely arranged, giving to it a light appearance. Nucleus usually also light.

(c) Parapyknomorphe Cells.—The arrangement is less dense than in a, denser than in b.

(d) Chromphile Cells.—Cells intensely stained, frequently quite homogeneous, looking like pigment masses, slender with sharp outlines and cork-screw like processes.

Only for one type of cells a name corresponding to the function can be given, viz., to the "motor cells" (which are found in the anterior horns, in all primary motor nuclei and in the "motor" regions of the cortex). The stained part of the substance of the cell body of these cells has a characteristic parallel striped arrangement which accordingly seems to be a characteristic for motor functions. By reason of the various arrangement of the stained substance of the cell-body the somatochrome cells may be classified as follows:

Group I. Arkyochrome Cells.—The stained substance is arranged in the shape of a network.

1. Type. Enarkyochrome Cells.—The lightly stained substance forms a net.

2. Type. Ampharkyochrome Cells.—There are two nets of stained substance, one formed by its intensely stained, the other by its lightly stained part.

3. Type. The Arkyochrome Olfactory Cells, etc.

Group B. Stichochrome Nerve Cells.—The stained part of the cell substance is arranged in the form of equally directed parallel stripes.

1. Type of the motor nerve cells.

2. Type of the large stichochrom cornu ammonis cells.

3. Type of the stichochrom cortex cells.

4. Type of the spinal nerve cells, etc.

Group C. Arkyostichochrome Nerve Cells.—Show parallel striped and network arrangement combined. The only type observed heretofore by N. as belonging to this group is the type of Purkinje's cells, etc.

Group D. *Gryochrome Nerve Cells*.—The stained part of the cell substance consists of minute granules (Körnchen). So far N. has not observed any type belonging to this group.

The nerve cells in which the nucleus forms the principal part are classified in the following manner :

(A) *Cytochrome Nerve Cells. Granuli*.—The cell-body is only rudimentarily developed (only traces of it present). The stained nucleus reaches the size of the ordinary leucocyte nuclei. Such cells are found in the granulous stratum of the cerebellar cortex.

1. Type of the A Granuli.—Cytochrome cells of the type a.

2. Type of the B Granuli.—Cytochrome cells of the type b, etc.

(B) *Karyochrome Nerve Cells. Nucleus Cells*.—The cell-body is rudimentarily developed. The stained nucleus has the size of nerve cell nuclei; in each case it is larger than the nuclei of the neuroglia. The cells of the *substantia gelatinosa* Rolando of the spinal cord belong to this group.

1. Type of the A Nuclei Cells.—Karyochrome cells of the type a.

2. Type of the B. Nuclei Cells.—Karyochrome cells of the type b, etc.

In each single type it would have to be noted whether the individual cell is *pyknomorphe*, *parapyknomorphe* or *chromophile*. As a rule, the *pyknomorphe* state corresponds to the relatively smallest volume of the cells, that is, the *pyknomorphe* cells are relatively the smallest, the *parapyknomorphe* larger, the *apyknomorphe* the largest.

N. inclines much to the view that the "*apyknomorphe*" state of the cell is that of rest, while the activity of the cell produces successively the "*parapyknomorphe*" and "*pyknomorphe*" states. At least he could state a considerable increase of "*pyknomorphe*" cells in the seventh nerve nucleus after faradic excitation. He emphasizes, however, that in all these the states cardinal structure which gives the cell its characteristic individual appearance remains unaltered.

The "*chromophile*" cells are still a puzzle. They are met with everywhere, although in much smaller number than the "*non-chromophile*" cells. Their significance is as yet unknown although it is certain that they are an "*inferior*" type. They vary in number and stay in an unaccountable manner. In stating pathological changes only those observations can, therefore, be made direct use of in which we know positively that they were not made upon *chromophile* cells. In all cases where it is doubtful whether the examined object is a *chromophile* cell or not, it remains uncertain whether pathological changes are present or not.

In other regards the stain allows such fine distinctions that the least pathological changes can be discovered. The study of the fine structure of the nerve cell is not made superfluous by Golgi's and Ramon y Cajal's methods. Only by a combination of both manners of investigations can we approach to full knowledge. Only those classifications based upon the cell connections as discovered by the methods of metal-impregnation which are in full harmony with the structural conditions of the nerve cells are justified scientifically.

The author refers to his article, "*Über die sogenannten Granula der Nervenzellen*" (*Neurologisches Centralblatt*, 1894, No. 13), in which the various types and states of nerve cells are illustrated by drawings.

ONCF.

#### PATHOLOGICAL.

*On the Lesions of the Crura Cerebri in Tabes*.—G. Pacetie, M.D. (*Rivista Sperimentale di Freniatria*, etc., 1894, page 518.) A report of one case, concerning a man of 40 years. A brother had suffered from convulsive fits, and two children were affected with eclampsia.

Patient had committed many sexual excesses. No indication of syphilis. Potation denied. Disease began in 1880, with lancinating pain in the last three toes of the left foot. At the same time he noticed diplopia and progressive impairment of vision, chiefly of right eye. The teeth of the upper jaw began to fall out without apparent cause, and without pain. Disturbances of gait chiefly in the dark. In January, 1893, following condition: Feeling of oppression and vertigo in head. Innervation of facial nerve normal, as is also that of the hypoglossus, aside from tremor in toto of tongue. Uvula deviates to the left. Mastication and deglutition normal. Bilateral paresis of the crico-arytenoidei post muscles. Sensibility to pain diminished over dorsum and sides of nose, over the superior alveolar process, hard palate, and in a lesser degree also over the whole region corresponding to the distribution of the V. nerve. Slight ptosis of left upper lid. Pupils unequal, do not respond neither to light nor accommodation. Aside from a slight mobility upwards and downwards, the eye movements abolished on both sides. No diplopia. Vision O. S. =  $\frac{1}{8}$ , O. D. = counting of fingers at distance of four inches. Lessening of sense of taste on the whole right side of tongue. Hyposmia of the right side. Upper extremities: Both sensibility, motility and muscular sense normal. Lower extremities: With open eyes no disturbances of station, gait, or of co-ordination of movements. With closed eyes vacillating station and gait, movements uncertain. Tactile and thermic sensibility aside from a slight impairment of localization well conserved. On lowest third of legs considerable hypalgesia, chiefly over dorsum and sole of feet, accompanied by retardation of the sensation of pain. Knee jerk and plantar reflex abolished on both sides. Repeated laryngeal crises, in one of which patient died in March, 1893. Microscopic examination of spinal cord and crura cerebri:

In the lumbar portion of the cord degeneration of the anterior, medial and posterior root zones (Flechzig); also of Lissauer's tract. In the dorsal portion degeneration in medial and posterior part of Goll's and lateral part of Burdach's columns. In the cervical portion rarification of Goll's columns. In the nuclei of the posterior columns rarification of the reticulum of nerve fibres.

Complete degeneration of both ascending V. roots up to the level of the crossing of the pyramids, further cephalad degeneration less pronounced.

Number of cells of the sensory nucleus propr. of the V. nerve diminished, reticulum of fibres rarified. Motor nucleus of the V. dorsal V. nucleus (Edinger), central path and radix descendens of the V. normal. Considerable atrophy of the substans ferruginea on both sides. Intense rarification of the solitary fascicles, but the arciform fibres which surround it on its dorsal part perfectly preserved. VI. nucleus perfectly atrophied without one normal cell, roots also intensely atrophic. The principal nucleus of the IV. deeply altered. Of the nucleus posterior (Westphal) no trace left. Fibres of trochlearis roots much altered, especially those of the right ones.

Moderate atrophy of the nucleus ventralis poster. (Siemerling) decreasing cephalad, more marked in the left than in the right one, in which latter it is quite insignificant. Intense atrophy of the whole nucleus dorsalis of the III., most pronounced in the nucleus dorsalis outer (Perlio). In the nucleus ventralis of the III. a larger number of cells, especially those situated medially, conserved, otherwise an atrophy which increases cephalad in intensity. Nucleus Edinger-Westphal. Altered in the same measure as the principal III. nucleus; the pars medialis better preserved than the pars lateralis. The nucleus medianus outer and nucleus bilateral. outer (Darkschewitsch) least altered. Of the root bundles of the III. those which take origin from the dorsal portion of the principal nucleus are missing altogether. The III. trunks

(nerves) highly degenerated. Longitudinal fasciculi of one posterior commissure entirely normal.

P. is much inclined to accept the view that the substantia ferruginea is connected with the V. nerve of the same side. The visceral crisis and laryngeal symptoms, also the disturbances of the cardiac innervation, have been caused by the grave alterations of the respiratory fasciculi.

The arciform fibre bundle which occupies the region dorsal from the respiratory bundle must be considered as a central path of the XII.; it may also contain a few fibres originating from the funic. gracil. et cuneat. nuclei, but in no way connected with the respiratory fasciculi.

As the IV. nucleus was affected in a higher degree than its roots, P. deems it very probable that the nucleus was primarily affected, the roots secondarily.

The nucleus ventralis posterior was nearly normal on the right, altered on the left side. Clinically there was ptosis of the left upper lid. P. concludes that the nucleus ventralis posterior represents the beginning of the III. nucleus and is probably the centre for the elevator of the lid; but it may also give off fibres for muscles supplied by the superior VII. nerve. The case throws no light upon the localization of the centre for the internal ocular muscles. P. accepts Schütz's and Siemerling's view that the pupillary reflexes are transmitted by way of fibres of the central pericavitary central gray matter. He found an intense rarification of the fibres of this formation, while Meynert's radial fibres, which Ross declares to conduct the said transmissions, and the posterior commissure to which Darkschewitsch describes said function, were perfectly normal.

ONUF.

**Pathogenesis of Acromegaly.**—Prof. A. Tamburini (*Revista Sperimentale di freniatria*, etc., 1894, fasc. iii-ix., p. 559).

The patient was an unmarried woman of thirty-six years. One brother had suffered for several months from a mental disease in which religious ideas predominated, and a sister was affected with melancholia. The patient's disease probably began at the age of twenty, and developed into typical acromegaly. She died from a progressive marasmus caused by intestinal catarrh. The autopsy revealed the presence of a tumor, size of a pigeon egg of the pituitary gland, affecting its epithelial part, while the "nervous" part was normal. No pronounced pathological changes of the thyroid gland. Brain without changes. In the spinal cord slight degeneration of Burdach's columns, and of the root zones of the cervical dorsal regions.

ONUF.

**On Traumatic Tabes and the Pathogenesis of Tabes in General.**—By Ed. Hitzig. Tabes is a chronic degeneration of the central nervous system—brain as well as spinal cord. Some neurological writers ascribe an ætiological importance to traumatism of any kind (mechanical or thermal injury to the periphery). Injuries to the skull and the spinal column, in so far as they are liable to be followed by gross anatomical changes of the brain and the cord itself, are not considered in the monograph.

In order to come to certain conclusions in the matter the author attempts to answer the following questions:

1. Is there an undisputed causal relationship between traumatism, (mechanical or thermal injury to the periphery) and the symptom-complex known as tabes dorsalis?

2. Are there, from an ætiological point of view, several different forms of tabes, or are they all dependent upon one common cause?

Our present knowledge supplies us with rather limited means for the solution of the first of the above questions. The literature on the subject being comparatively meagre and partly incomplete, we hardly can expect the result of this research to be conclusive. Four cases of the author's personal observation, 41 cases scattered through the older litera-

ture, and 21 unpublished cases of Erb form the material. Out of this totality of 66 cases, 10 or 11 cases only meet the requirements of sound criticism. The evidence thus afforded justifies, in the author's opinion, the following conclusion: The occasional onset of the disease in the injured side, or even the permanent preponderance of the symptoms on this side, are neither constant nor exclusive phenomena as to enable us to construe a clinical picture characteristic of the traumatic origin of the disease. In spite of the great importance previous venereal infection plays in most of the cases of tabes, we meet with cases showing a distinct relation to traumatism, and which cannot be explained on any other grounds.

The three succeeding chapters of the monograph are devoted to the solution of the second of the above questions.

The author asserts that, in his opinion, tabes never takes its origin from a peripheral neuritis invading, as an ascending degeneration, the cord by the way of the posterior roots. The most common anatomical findings in cords of tabics are degenerations in the posterior roots and columns—both being in corresponding quantitative relations. As proof of these views the author cites the results of anatomical researches made with that object in view by one of his assistants (Dr. Wollenberg: *Untersuchungen ueber das Verhalten der Spinal ganglion bei tabes dorsalis Arch. f. Psychiatric*, vol. xxii, p. 313.) Dr. Wollenberg found in all examined cords, even in those with extensive lesion in the posterior roots, the peripheral nerves originating from the intervertebral ganglion, and the latter itself perfectly normal. The disease might invade slightly the cells of the ganglions, not interfering with the performance of its function.

These facts, in conjunction with the Wallerian law of degeneration, hardly admit any other explanation than the following: The degeneration of the posterior roots met with in cases of typical tabes, cannot be looked upon as a descending degeneration towards the spinal ganglion. In all probability it is an independent, and most probably the primary process, from which the degeneration of the cord arises, partially, at least, as a secondary ascending degeneration. On account of the discrepancy, though existing sometimes between the extension of the pathological changes of the root-fibres in their intra- and extra-medullary course, to the above statement, the addition has to be made, that the irritation of the posterior roots destroys the whole course of the fibres from the inter-vertebral ganglia. Under the influence of unknown circumstances, occasionally root fibres stay temporarily or permanently unaffected, the intra-medullary part of which is degenerated.

According to P. Marie, the nerve fibres, not having an anatomical independence, cannot be affected primarily by disease. In cases of tabes, an affection of the sensory fibres, the origin of the disease must be looked for in their nutritive cells—the cells of the inter-vertebral ganglia or cells of similar physiological dignity, scattered probably in the periphery. This theory is not proved sufficiently. Moreover, recent investigations show that after lesions of the peripheral nerves (amputations, neuritis, etc.) the cord is found to be diseased. But no case has yet been reported in which the familiar anatomical or clinical phenomena of tabes have been observed.

It is commonly known that tabic symptoms result from lesions in parts of the nervous system, which are neither anatomically continuous nor physiologically related. Traumatic tabes does not show any features distinguishing it from tabes following other causes. It is, therefore, evident that the trauma must affect all the parts of the nervous system that are affected in common forms of tabes.

Undeniably syphilitic infection is the most prominent ætiological factor of tabes. But all statistics agree that about ten per cent. of the cases are not attributable to this cause. Under the heading, syphilis, the

authors consider every venereal infection; but not every venereal infection is necessarily syphilitic. Furthermore, it is a familiar observation that just the milder forms of infection, cases without or very mild secondary symptoms, are mostly liable to be followed by tabes. Lastly, the pathological findings in tabes are quite unlike all the other pathological presentations of the syphilitic infection. With all this the percentage of non-syphilitic cases of tabes gets yet larger.

According to Strumpell the pathological character of tabes is allied to degenerations following other acute or chronic infections or intoxications. These degenerations, too, do not prevent the characters of the original exciting disease. Through the influence of the infection, chemical poisons arise, which, like others, show distinctly elective qualities.

With the aid of this theory we vainly attempt to explain the pathology of the cases not having any relation with syphilis.

Unitarians and dualists are both wrong. The author believes that venereal infections in general, and especially syphilitic infection, are caused by several poisons, either vaccinated at the moment of the infection, or created later in the course and through the development of the disease. One poison causing the primary sclerosis, might lead to secondary and tertiary symptoms; another poison, acting either at the moment of the infection, or originating later, results in a morbid disposition—changes in the blood. After a short or longer interval of time, spontaneously or after a slight exciting cause, a degeneration of the nervous system might set in as a sequelae of the morbid condition left behind. Degenerations of the nervous system caused by ergotism or pellagra are analogous phenomena.

If we can prove that trauma or refrigeration is liable to produce similar changes themselves or fertilize the organism for the action of the supposed poison, traumatic tabes can be pathologically understood.

Unless this is proved tabes following traumatism is yet an open question.

FRAENKEL.

#### CLINICAL.

**Glio-Sarcoma of the Cerebellum.**—P. de Michele (*Annali di Neurologia*, 1894, p. 227).

A male, 16 years old; no syphilis; no alcoholism; no tobacco. One aunt epileptic. In January, 1892, the patient had an attack characterized by general fatigue, slight headache and slight fever, lasting about four days, which was called "influenza" by the attending physician. A week afterwards he began to have attacks of vomiting. They occurred before breakfast; and were accompanied by headache. These symptoms were associated with dyspepsia, exhaustion from the least amount of work, pain in the upper part of the dorsal region and pain in the upper extremities with violent exacerbations, which were relieved temporarily by the cautery applied to neck.

Further, the patient uttered loud yells without noticing it. Later, pain in the lower extremities, the headache localized itself in the parieto-temporal region of the right side. At this time examination revealed sensorium free, legs normal, innervation of face normal. Strabismus convergens of left eye. Bilateral neuritis optica with beginning atrophy of disc. Inco-ordination of movements of the tongue. Sense of taste on right side of tongue impaired in the anterior part. Scoliosis of the whole vertebral column with convexity to the right; head inclined to the left so that the whole body formed a curve with convexity to the right. The muscular power of all extremities diminished, muscles show lessened response to galvanic and faradic currents. General impairment of nutrition, especially marked in upper extremities. In the last months before death, sense of hearing impaired on both sides. Shortly before death paraplegia without sensory disturbances. Date of death not stated. The autopsy revealed the presence of a glio-sarcoma at the base of the

brain, in the thalamus optici, velum posterior, and chiefly in the vermis inferior cerebelli. The neoplasm gave the character of a gradual invasion into the nervous substance, forming small groups of the pathological cellular elements, from which again noduli of various sizes are formed.

The author concludes that :

1. The glio-sarcomata can clinically manifest themselves by a course of disease similar to that of a chronic inflammatory process.
2. Their clinical manifestation and their histological structure make it probable that they are of infectious origin.
3. The most important diagnostic symptoms for localization of a process in the vermis inferior of the cerebellum are: (a) lateral curvature of the vertebral column; (b) progressive weakness of the extremities; (c) general hypotrophia; (d) unconscious yelling or screaming.

ONUF.

**Treatment of Epilepsy.**—Moeli (*Therapeutische Monatshefte*, September, 1894).

In patients in whom the course of the epilepsy is not benefited by the use of bromides the author has used atropine for the past three years. The number of cases in which he has used it is thirty-seven, of which four were excluded as not being cases of genuine epilepsy. The dose of atropine,  $\frac{1}{120}$  of a grain twice a day, is kept up for a period of from seven to eight weeks. During the administration of the atropine the bromides are continued. In about a third of the cases the beneficial effects of the combined therapy could be seen both in the decrease of the number of the attacks and a lessening of their severity. In two women and three women the attacks were stopped for several weeks, although previous to that time a much larger daily quantity of bromide had not succeeded in causing so good a result.

J. C.

**Notes on Hypnotics.**—McCraig, (*Med. and Surg. Reporter*, February 16, 1895).

McCraig reports a number of cases, including acute delirious mania, chronic melancholia, acute melancholia and approaching climacteric, in which the absence of sleep called for remedial measures. He obtained good results with sulfonal (20 to 60 grains) and trional (15 to 40 grains). In two cases in which sulfonal had been administered in doses respectively of 60 and 30 grains, retention of urine followed, necessitating catheterization. Craig considers these drugs, in some measure, ideal hypnotics.

MEIROWITZ.

**Gold as a Therapeutic Agent.**—Walling (*Med. and Surg. Reporter*, January 26, 1895).

The author refers to Gobert, Féré and Shtcherback as finding the bromide of gold decidedly more powerful in its effects upon the nervous centres than the bromides of sodium and potassium. Combined with arsenic, the bromide of gold exerts its profoundest effect. Waller has used in locomotor ataxia and kindred affection a solution containing the tribromide of gold and the oxybromide of arsenic, ten drops of which represented  $\frac{1}{32}$  of a grain of the tribromide of gold and  $\frac{1}{32}$  of a grain of the oxybromide of arsenic. In several cases of epilepsy this solution has proved to be more efficacious than any other drug. Walling finds it useful in the fibrillary twitchings of some spinal affects: it restores tone in nervous condition, and has a most marked effect on rheumatism. In doses of seven to ten drops, it is beneficial on sexual debility. It also acts well in sciatica and other neuralgias.

MEIROWITZ.

## Society Reports.

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### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, held at the New York Academy of Medicine,  
on February 5, 1895.*

Dr. C. A. Herter, Vice-President, in the chair.

### CHARCOT MONUMENT FUND.

DR. HERTER, Chairman of the Charcot Monument Fund Committee, reported that \$533 have been received. This amount was mostly made up of large subscriptions, and there has been a lack of general interest in the matter. The committee hoped to increase the amount to \$800 or \$1,000, so as to make a really worthy contribution to the general fund, and in order to accomplish this, it was very desirable that each member of the society should contribute something, no matter how small the amount.

### ATHETOSIS WITH BEGINNING TABES.

DR. JOSEPH COLLINS presented a male patient, thirty-seven years old, married, and by occupation a policeman. The family history was very good. Syphilis and alcoholism were denied, and there were no traces of either to be found. The man had never used tobacco nor indulged in excesses. The only factor in his previous history that was at all pertinent was that nearly eight years before coming under observation, while attempting to make an arrest, he was thrown against a pillar of an elevated railway, and received a severe cut over the right parietal region. He was not unconscious, but was incapacitated for about three weeks. There had been no headache since then.

For a year there had been noticed some involuntary



movements of the right hand, particularly on excitement and exertion. Accompanying this was a sense of numbness in the hand and upper right extremity, and a loss of dexterity in the fingers of this hand. There was also drooping of the left eyelid, at first intermittent, but subsequently constant. Further, transient attacks of diplopia occurred. The numbness of the right hand and arm gradually grew worse, and the member felt heavy and useless. For a short time a similar feeling of numbness had been perceived in the legs, particularly in the left, together with the sensation of a firm band fastened around the calf of the right leg and about the chest. Sexual vigor had begun to diminish within six or seven months, until gradually a state of impotency was reached. There was no trouble with the sphincters, and there had been no disturbance of speech and but slight uncertainty of gait, either by day or by night.

Examination showed more or less irregular movements of the right upper extremity, particularly manifest in the hand. These movements constituted neither a clonus nor a tremor, nor were they sufficiently rhythmic, determinate, systematic, or uniform to be called strictly athetoid, and there was no tendency to distortion. They continued during sleep, but to a less degree, and were aggravated by stimulation and excitement. They consisted in a sort of flexion and extension of the hand on the wrist and irregular movements of the fingers. They could not be diminished by the strongest endeavor on the part of the patient. The strength of the right arm was a little impaired, more in the extensors than in the flexors. There was considerable tactile anesthesia, but the pain-sense and thermal sense were normal. The muscular sense was impaired, and there was loss of the sense of position in the right arm. When a coin, such as a quarter-dollar or half-dollar, was dropped into the palm, the man could feel it strike, but he had no idea what it was, nor could he tell when it was removed. There was marked ataxia in this extremity, complete loss of dexterity in the fingers, and great overaction on attempts at directed effort. The left palpebral fissure was narrowed, and there was very slight real ptosis; the left eye seemed small and retracted, while the pupil was pin-head in size and did not react to light and very slightly in accommodation; there was slight muscular deficiency of the left superior rectus. The pupil of the right eye was small; it reacted in accommodation, but

not to light. There was no limitation in movement or of the visual field in either eye. Both optic nerves were pale, the left rather more so than the right. It was thought they were possibly in the first stage of atrophy. Vision, R. 20 L; L. 20 XL; color-perception was good; there was no scotoma.

The knee-jerks were completely absent, even with reinforcement. Station was moderately steady. There was no twitching of the left hand, but the fingers had lost their dexterity to some extent. There was slight numbness in the left upper extremity, but objectively sensibility was normal. There was no muscular atrophy. Although the patient strenuously denied syphilitic infection, and there was no evidence of the disease, he was put upon mercurial inunctions and increasing doses of potassium iodide. This treatment, combined with warm baths, rest in bed, massage and tonic, was kept up for ten weeks, and at the end of that time the patient had not shown any marked improvement, excepting the return of sexual potency. The treatment was then changed to gold and arsenic chlorid, the iodide being kept up.

Dr. COLLINS regarded the case as one of athetosis, with beginning tabes. This combination is rather rare, only a few cases having been reported. The only symptoms of tabes in this case were the loss of knee-jerks, the condition of the man's eyes and the impotency. He had never had any pains.

Dr. M. ALLEN STARR said the case impressed him as one of multiple lesions, and in the absence of pains, together with the marked unilateral character of the symptoms, it differed very decidedly from locomotor ataxia. A person may have ataxia without necessarily having locomotor ataxia, and it may be the result, as may also the loss of knee-jerks, of a lesion in the pons, or crus, or cerebellum.

Dr. B. SACHS said that on account of the multiplicity of the man's symptoms he did not regard the case as one of locomotor ataxia, pure and simple. He rather suspected that the case was one of cerebro-spinal syphilis. He expressed the opinion that one of the most certain symptoms in cases of syphilitic tabes—as distinguished from pure tabes—is an inequality in the pupils; either they are unlike, or they do not act alike.

Dr. STARR said that he felt inclined to agree with a statement made by Dr. Buzzard, of London, in a recent book, that multiple sclerosis is a much more frequent

disease than has heretofore been supposed. That writer reports five cases of multiple sclerosis in which the ordinary diagnosis would be locomotor ataxia.

Dr. COLLINS said that he regarded the case as one of cerebro-spinal syphilis of the tabic type. The patient was seen a number of times by Dr. C. L. Dana, who made the same diagnosis. Dr. Collins added that he did not think it possible to have a localized lesion of the crus or cerebellum without the development of certain motor and other symptoms which this man did not present. This man probably had syphilis many years ago, and there was now some thickening in the capsule that gives rise to the athetosis. The tertiary or quartan manifestations of the previous syphilis were beginning to be manifest in the posterior roots and columns of the cord and the result clinically is the taboid symptoms.

Dr. HERTER said that he thought the case was one of multiple sclerosis rather than cerebro-spinal syphilis. He did not consider it one of locomotor ataxia. It is perfectly possible to have a lesion of the crus that gives rise to anesthesia and inco-ordination on one side. In many cases of multiple sclerosis one of the early symptoms is slight optic atrophy.

#### A CONTRIBUTION TO THE SUBJECT OF TUMORS OF THE SPINAL CORD, WITH REMARKS UPON THEIR DIAGNOSIS AND THEIR SURGICAL TREATMENT, WITH A REPORT OF SIX CASES, IN THREE OF WHICH THE TUMOR WAS REMOVED.

Dr. M. ALLEN STARR stated that the diagnosis of tumors of the spinal cord has only become possible with any degree of accuracy within the past few years, since the localization of the motor and sensory functions of the cord has been precisely determined. The practical importance of a subject of this character cannot be exaggerated, inasmuch as a study of the cases upon record shows that relief of the symptoms and cure of the disease are perfectly possible by surgical interference in the majority of cases when the diagnosis is made in time.

From a careful review of the literature of the past ten years he has been able to collect 123 cases of tumor of the spinal cord. In 100 of these the history was fairly satisfactory, and in the light of our present knowledge a diagnosis should have been quite clear some time before

death. In fifty-four of the cases it should have been possible to reach not only a diagnosis, but also a conclusion as to the feasibility of an operation. Studying the cases from a pathologic standpoint, it was found that of the 100 cases there were seventy-five in which the tumor could have been removed. As a matter of fact, operations had been undertaken for the relief of tumor of the spinal cord in twenty-two cases (not included in the 123 cases previously referred to). In only two instances had the surgeon failed to find the tumor sought for; in but one was it impossible to remove it when found. In eleven cases the patients died soon after the operation; in eleven cases they recovered from the operation. In six cases the operation was followed by recovery from the condition of paraplegia. After fully reporting six cases of tumor, in three of which the diagnosis was made and the tumor successfully removed, Dr. Starr said that the most important and earliest symptom of tumor of the spinal cord is pain. The location of this pain is usually in the peripheral distribution of the nerve-root that is first compressed by the growth of the tumor. In the majority of cases the pain is referred to the epigastrium or to the abdominal region, or to the legs or arms, rather than to the spine itself. The pain is of a severe neuralgic character, is sometimes described as burning; and, as it increases, it appears to shoot along the course of the nerve that is implicated. The second group of symptoms of diagnostic importance are those caused by the compression of the cord by the tumor. It is well known that when compression is exerted either upon a nerve-trunk or upon the spinal cord, a certain order is commonly observed in the symptoms produced, viz: (1) pain; referred to the periphery; (2) increase of reflex activity; (3) paralysis; (4) loss of sensibility; (5) loss of reflex activity. In the vast majority of cases tumors of the cord have been found in the dorsal region.

A summary of the cases collected from the literature of the past ten years showed that thus far the number of recoveries after the removal of spinal tumors has been but six, and in one of these the compression was exerted upon the cauda equina and not upon the cord proper. It must be said in explanation of these unfavorable results that the operation was not undertaken until the tumor had been present for a number of months, and until the secondary destruction of the spinal tissue was of such a nature that regeneration of the nerve-elements was practically out of the question.

In spite, however, of the gravity of the situation, and of the very unfavorable results that have thus far attended the removal of spinal tumors, it is always to be remembered that the disease is otherwise of a hopeless character and of a necessarily fatal termination; and hence any measures for its relief are justifiable. It is also to be remembered that the earlier the diagnosis can be undertaken, the more likely it is to be followed by relief.

Attention was called to the relation of the segments of the spinal cord to the vertebræ, and it was pointed out that tumors lie from two to four inches above the line of anæsthesia on the body. Large incisions, the removal of the bone by cutting forceps and rongeur, the cautious division of the dura, and care in handling the cord to prevent hæmorrhage and pressure were recommended.

Dr. C. L. DANA said that cases of spinal tumor have been rare in his experience. He recently saw a case in which he felt inclined to make that diagnosis, although he could not say positively whether the lesion was a tumor or not. The patient was a female, aged thirty years, who was operated on about a year ago for carcinoma of the foot, the leg being removed above the knee. The patient made a good recovery, and remained well until about nine months after the operation, when she began to suffer from neuralgia on the right side, in the region supplied by the seventh and eighth dorsal nerves. She gradually grew weaker, and there was a certain amount of cachexia. Four months after the onset of the pain paraplegia developed, which, within a week, became complete for both motion and sensation. There was a loss of control over the sphincters, and the woman presented all the symptoms one would expect from a transverse lesion of the cord. The sudden onset of the paraplegia made it probable that in this case there was a malignant growth of the cord, with, perhaps, acute softening due to hæmorrhage or embolism. Dr. Dana added that thus far the result of the surgical treatment of tumors of the cord is rather discouraging; perhaps it will be better in the future, when the tumors are recognized earlier.

Dr. SACHS said that this subject evidently possesses a greater interest now than it did a number of years ago when Erb made the statement that spinal tumors were of so little importance that he did not care to enter into

their differential diagnosis. It is doubtful, however, whether the practical importance of the subject will, in years to come, prove to be much greater than it has been in the past. In most of the cases reported as operated on with good results the tumor was extra-dural. In the diagnosis of these cases it is a point of the greatest importance to try and make out whether the growth is extra-dural or intra-dural. Further, it is important to differentiate between the extra-spinal and intra-spinal. When the cord itself is involved, it is very doubtful whether anything can be gained by an operation.

Dr. Sachs said that, while he was perfectly willing to acknowledge the value of pain as a means of diagnosis in these cases, he thought Dr. Starr had laid rather too much stress upon this. The pain is produced by tumors that rest chiefly on the posterior surface of the cord, and, while undoubtedly a large number do take their origin in that region—perhaps the majority—still there are some, particularly those involving the vertebræ that would be likely to give rise to symptoms referable to the anterior nerve roots rather than to pain. These so-called root symptoms are often very important. Before advising an operation in these cases, it is well to try to arrive at a conclusion as to the character of the growth. In cases in which there is any suspicion of tuberculosis, it is doubtful whether one should operate; also in specific cases. The operation is of so serious a nature that everything must be carefully considered before it is resorted to. Dr. Sachs called attention to the fact that some cases in which the symptoms might very well lead one to suspect tumor might prove to be cases of localized specific meningitis. Such instances have come under his observation. Thus far, the results following spinal operations have been extremely discouraging, and the prospects are even more gloomy than with brain tumors, or fully as much so. After operations on the spinal cord, the patients, for some reason which has not been determined, do not seem to improve. This is probably due to structural changes in the cord, so slight in extent that they are not noticeable to the naked eye.

Dr. A. J. McCOSH discussed the subject from a surgical standpoint. He stated that at the present time the outlook in this field of surgery is rather gloomy. One obstacle to the attainment of brilliant results lies in the character of the growths. The great majority of spinal

tumors seem to be malignant and often tuberculous, and in either case very little can be expected from their removal. The operation of laminectomy, *per se*, is not a formidable one; it is rather tedious, and considerable blood is likely to be lost; but if the patient is in fair condition the mortality should not be great. His own results with the operation have not been encouraging. He has operated four times, and in none of these cases did any benefit follow the operation. Still, it must be remembered that these are otherwise hopeful cases, and if an operation offers a reasonable hope for relief it should be undertaken.

Dr. HERMAN M. BIGGS said that in the cases of malignant tumor of the cord that he has seen at autopsy there were invariably similar growths in other parts of the body and secondary tumors in the brain. The only kind of tumor of the cord in which the prognosis is at all favorable is the non malignant connective-tissue neoplasm, such as has been removed by Dr. Macewen. In cases in which the growth is tuberculous or malignant the course of the disease is probably much more rapid than when it is non-malignant, and this fact might give some clue as to the character of the tumor and the advisability of operative interference.

Dr. HERTER said that he had seen a number of cases of tumor of the spinal cord, and has recently reviewed the literature of the subject. In doing this he was impressed with the idea that the time had not yet arrived for writing the history of spinal tumors. A sufficient number of these cases have not yet been accurately reported. Dr. Starr has done great service in calling attention to the sensory disturbances met with in these cases and by his careful study of them. He fully agreed with Dr. Starr that pain is the most constant and characteristic as well as the earliest symptom of tumor of the cord; it is much more constant than in brain-tumor.

As regards the character of these growths, Dr. Herter said that of the cases thus far reported carcinomata do not form a very large proportion. The sarcomatous and tuberculous are more frequent, and many of them begin in the spinal cord itself. The gray matter of the cord seems to afford a favorable nidus for the growth of tubercle-bacilli. Probably one-half of the tumors of the cord will never be classed as operable.

Dr. STARR, in closing the discussion, said that he had thus far been unable to find any definite points of dif-

ferential diagnosis between extra-dural and intra-dural tumors of the cord. There are some points of differentiation between the intra-spinal and the extra-spinal, including under the latter heading both the intra-dural and extra-dural. With the intra-spinal there is more likely to be an early development of bedsores and cystitis; also reaction of degeneration and atrophy of the muscles. The supposed rarity of these cases is probably largely due to the fact that they are often unrecognized. The same is true of other diseases. Eight years ago syringomyelia was rarely met with; since that time the disease has been studied and well described, and last year sixty-eight cases were reported. Dr. Starr said that within a year his paper on spinal tumors will no doubt be outgrown, but he expressed the hope that it would lead to a more careful study of these cases. The statistics thus far are not so discouraging, and there is every reason to hope that they will be better in the future. Of twenty-two cases thus far operated on, six recovered from the operation and the paraplegia—that is, more than twenty-five per cent. It is true that in most cases the character of the tumor is such that recurrence is to be feared; still, surgeons do not hesitate to remove a sarcoma in other parts of the body, even though the probability is that it will recur.

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## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, January 28, 1895.*

Dr. WHARTON SINKLER, President, in the Chair.

Dr. PHILIP COOMBS KNAPP, of Boston, read a paper entitled

### THE ALLEGED REFLEX CAUSES OF NERVOUS DISEASES.

#### DISCUSSION.

Dr. WILLIAM OSLER, of Baltimore.—With most of Dr. Knapp's conclusions I am entirely in accord, more particularly with the remarks upon the relation of uterine



and ovarian troubles with the various neuroses alleged to be excited by disease of these organs.

There are, however, several conditions which we cannot, with our present knowledge, explain except on a theory of reflex influences. I refer more particularly to some of the curious phenomena connected with adenoid vegetations in the pharynx and disease of the turbinated bones, such as persistent asthmatic attacks in children, which are cured radically and permanently by local treatment; also similar spasmodic affections of the bronchi in adults. I have recently had one or two striking instances of this condition. Eighteen months ago I saw a boy of eleven years who since the age of eight years had had recurring attacks of asthma of such severity as to produce changes in the form of the thorax. Complete and thorough treatment of the naso-pharynx and turbinated bones was advised, since which the boy has not had a single attack.

Then we have the fact of a few well authenticated cures of exophthalmic goitre by treatment of the turbinated bones.

Of course, I think that a great deal of twaddle has been written with reference to the influence of eye strain in producing epilepsy and chorea. Among the cases of chorea submitted to Dr. Stevens, and the cases reported by Dr. Ranney, there is scarcely a case of genuine Sydenham's chorea. They are cases of habit spasm. That such cases are cured by the removal of nasal and pharyngeal trouble is undoubted.

On the question of headaches, Dr. Knapp takes, I believe, an extreme view. I think that a larger percentage of cases of headache are cured by removal of ocular defects than he is willing to allow.

Dr. GEORGE E. DE SCHWEINITZ.—As one of my distinguished confreres has reviewed certain writings of mine with reference to eye strain with this comment, "It may suit the general practitioner, but it is not in accord with the experience of ophthalmologists," or words to that effect, I presume I am safe in saying that I should not be accused of too great partisanship on the side of ophthalmic medicine. I am in entire accord with Dr. Knapp in the belief that Sydenham's chorea is not caused by hypermetropia and hypermetropic astigmatism, or, indeed, by any ocular anomaly, refractive or muscular. As I have elsewhere written, "the evidence seems quite as lacking that hypermetropic refraction is the basal

cause of chorea as it is that the chorea is the cause of the hypermetropia." In a constitution predisposed to chorea I presume eye strain is an important factor in fostering and provoking attacks. Pseudo-choreas are often the result of anomalies of refraction.

That true epilepsy is ever produced by refractive anomalies I doubt, although I am quite sure that convulsive seizures of various types, and no doubt some of those which belong to epilepsy proper, are modified and sometimes checked for long seasons of time by the use of proper glasses, prismatic or sphero-cylindrical. The great difficulty resides in the fact that the reports of these cases have been rushed into print before the proper time has elapsed to test the permanency of the effect. The evident conclusion of the matter is that while we may not believe the extravagant assertions that have been promulgated with reference to the effect of eye strain, we do know that in the management of functional nervous disorders it is one of the influences that must be subdued before the treatment of the case is successful, for precisely the same reason that the defective functions of any other organ should be put into proper order in the management of these cases.

I am not in accord with Dr. Knapp in his estimate, as I understand it, of the value of correcting refractive errors in the treatment of headache. In the first place, I do not believe that the cause of headache can ever be inferred from the position of the pain. The patient may experience the painful sensations from eye strain in any portion of the head—frontal, parietal, occipital or vertical region. Again, the pain is often situated between the shoulder-blades, far down in the neck, and sometimes over the præcordium. It is a notorious fact that in many cases of the most pronounced "eye strain cases" the eyes themselves and their immediate vicinity have been entirely free from pain. I do not believe that there is the slightest doubt, other things being equal, that fully sixty per cent of functional headaches will be materially benefited, or cured, by the proper correction of refractive anomalies.

In regard to the frequency of the various types of refractive error, it should be remembered that the earlier statistics are misleading, but owing to the beautiful statistical work of Dr. B. Alex. Randall, of this city, and careful records, such as have been made by Dr. Risley and other observers, we are in a position to state the

matter with reasonable accuracy, and know that hypermetropic refraction is the preponderating condition in childhood, being found in seventy-six per cent of the eyes of children in the elementary schools.

There is one very important point in the management of many of the cases of neurasthenia, in which, sometimes, the correction of refractive error, although evidently indicated, is not followed by good results. These cases, with bad nervous ancestry, if they are also handicapped by an anomaly of refraction, always work, as everyone does who is so handicapped, at serious disadvantage, only in them the nervous system receives the heavier impress. Months and even years go by, and the impress upon the nervous system is deeper and deeper. Now the refractive anomaly is corrected, but we cannot hope by this alone to remove the long standing results which have been practically seared into the system. I can best illustrate this by the case of a young woman from the South, a patient of Dr. Weir Mitchell, who came to him for most atrocious headaches, as well as other symptoms of a pronounced neurasthenic type. Sometimes her headaches were so bad that, to use a common expression, she almost "went into convulsions." She had a high refractive error and considerable muscular defect—the so-called esophoria. She was put to bed, and underwent the ordinary rest cure for a time, with little or no benefit. Then, at Dr. Mitchell's request, the refractive and muscular anomalies were corrected absolutely by means of glasses and tenotomies. Again there was little improvement. Once more the patient was put to bed and underwent a second rest cure, with entire relief, my remembrance being that in about six weeks she was entirely free of headaches, and has been for many years a healthy girl. This illustrates the method which should be pursued in these cases, and it, moreover, illustrates that while, no doubt, many of the neurasthenic tendencies and symptoms are hereditary, in a disposition of this type eye strain long continued may precipitate and, in that sense, cause the break down, precisely as it may bring about headache and other phenomena with which we are all familiar. I am quite sure that we all agree, and I think Dr. Knapp has said, that in the treatment of all of these cases the examination of the eye is of very great importance, precisely as is the examination of the nose, or, indeed, of any other organ, in order to make sure that the investigation of the case has been a perfect one.

Dr. A. K. MOULTON.—I do not believe that very many cases of insanity are caused by uterine disorders. In the service of a large hospital, in which a woman was employed, very careful examinations were made of four hundred women. The physician had previously had experience in caring for pauper women supposed to be mentally sound, and she discovered actually fewer cases of uterine disorder among the insane than among the sane. I have myself seen very few cases of insanity in women which I believed to be due to disease of the reproductive organs.

Dr. GUY HINSDALE.—I recall two cases of epilepsy due to peripheral irritation. One case was reported to this Society by Dr. Brubaker, in which carious teeth were ascertained to be the cause of epilepsy. After their removal the epilepsy disappeared and has remained absent seven or eight years.

The second case I saw at the Infirmary for Nervous Diseases. A sailor had received, among other injuries, a severe blow on one testicle. In six or seven weeks this was followed by spasms which were repeated at frequent intervals, until they reached twenty-five in a day. The diseased testicle was removed, and from that day he has not had another attack during the six years that have elapsed. If in cases of epilepsy we look for peripheral causes we shall more frequently find them. We are too apt to neglect a detailed investigation of cases of epilepsy.

Dr. G. BETTON MASSEY.—Dr. Knapp makes an important reservation in treating of gynecological vagaries in relation to the reflex origin of nervous disease, when he speaks of the possibility of distinct disease in the pelvis giving rise to referred pain or neuroses. It is operation in the absence of such distinct disease that constitutes the worst vagary that has attacked the profession. The fact that for years the profession assumed that the normal ovaries could produce nervous disease, was probably the source of the attack on these unoffending organs. If the organs were diseased we could easily see that they might have the same effect as a carious tooth; like the carious tooth, removal is not the only remedy.

I think that it is a pity to allude to the ovary so much in speaking of these questions. The uterus is a far more important source of irritation. Diseases of the uterus, especially catarrhal diseases, are a very important source of referred pain in the back, head and spine, and of general neuræsthenia.

Similar conditions occur in catarrhal inflammation of the male pelvis. I have seen a number of cases of prostatitis with profound nervous symptoms which were quickly cured by relief of the prostatic condition.

Dr. FRANCIS X. DERCUM.—I am one of those who believe in the value of treatment of the eye in cases of headache. I am also one of those who are prejudiced against operative interference with the pelvic organs in these cases, for I have seen the nervous symptoms not only continue, but be aggravated after such treatment and even new symptoms be developed. This is, perhaps, not remarkable when we consider that in the removal of the ovaries or uterus, nerves must be injured and changes may take place analogous to those sometimes met with after amputation.

This discussion might be continued much longer without adding much that is new. I am a confirmed believer in the doctrine of referred pain, but the actual occurrence of reflex neuroses has diminished in my personal experience with my own advance in knowledge of nervous troubles.

Dr. J. MADISON TAYLOR.—I cannot refrain from alluding to two ancient beliefs which have come much to my attention among children's ailments, viz., the adherent prepuces and lumbricoid worms, as a presumed source of much reflex disease. In the matter of the adherent prepuces I have steadfastly set myself against this greatly exaggerated trouble for fifteen years. I have seen no grave instance of disturbance from this cause alone, although it amply merits attention always when present, but circumcision is rarely needed; only judicious stripping such as I have constantly taught.

Lumbricoid worms were a most convenient explanation of the phenomena of motor excitements, as well as the chronic intestinal disorders which have failed to receive due attention when once the worms were demonstrated and exercised. But what has become of the *ascaris lumbricoides*, or even the *oxyuris vermicularis*? I hear of them so rarely and seldom meet them even in a very large dispensary and other practice. Last winter one colored boy presented himself with fair health but huge belly, from whom we dislocated 366 large round worms. These cases are most rare now.

Dr. PHILIP COOMES KNAPP.—As to Dr. Massey's suggestion that actual disease of the ovaries or other pelvic organs may give rise to nervous disease. I would say

that he has been called a poor pathologist who cannot find some pathological condition in an ovary that has been removed. In most cases of "normal ovariectomy" there has been said to be some slight process of sclerosis, or a small cyst which was thought to cause the trouble and to justify the operation. The point which I wished to make was that unless there are distinct symptoms of disease of an organ sufficient to attract attention to that organ, it is not likely to cause any remote or reflex disorder. Where there is a history of long continued pain or where there are inflammatory processes, etc., then the nervous phenomena are entirely secondary, and the etiology is different from that of a reflex neurosis.

There is, perhaps, not so much disagreement between my position and that of Dr. De Schweinitz as he thinks. I think that I said that I make it a routine practice to refer almost every case of headache to the oculist, and I rather pride myself that in some instances I have held firmly against the opinion of the oculist that the headache was due to eye trouble, and the subsequent history has shown the correctness of my view. I have gradually been coming to the belief that one of the distinctive symptoms in refractive headaches is the aggravation of the pain by the use of the eyes for near work. Another symptom that seemed to me to be important, although not so conclusive, is the location of the pain. With the routine practice of referring every case to the oculist, I have obtained satisfactory results in a great many cases, but there is still a considerable remainder of cases where treatment of the eyes had not cured the headache.

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**Original Articles.**

REPORT OF TWO CASES OF CEREBRAL  
TUMORS MISTAKEN FOR PARESIS, WITH  
AUTOPSY.

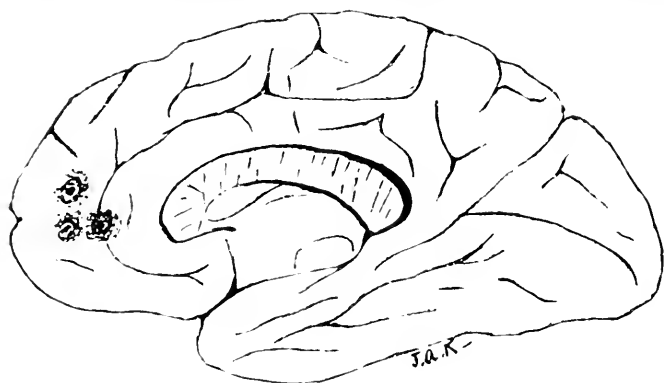
By L. PIERCE CLARK, M.D.,  
Middletown, Conn.

CASE I.—Syphilitic gummata of the brain. Name R. P.; nativity, Italy; 49 years old; married and a shoemaker by trade. His habits and the cause of his attack were unknown. When admitted to the asylum, December 12, 1889, he was dull and stupid, simple and childish. "He did not appreciate his condition or surroundings and could give no satisfactory account of himself. A physical examination made upon his admission proved to be entirely negative. The patient continued quiet and depressed, showing no well marked delusions, but complained of considerable pain in the head over the frontal region, which persisted more or less throughout the remainder of his illness, death occurring January 25, 1894, while in a convulsion.

During the entire illness, the patient was depressed, except on two occasions, when he expressed himself as feeling "all right" and "good." Three years after admission, his condition was one of extreme dementia. His gait was ataxic and he could pronounce simple words only with great difficulty. The tongue, lips, facial muscles and fingers displayed marked muscular tremor. The knee-jerk was very much exaggerated, but was equal on both sides. Three months before death, he had several

convulsions each lasting from five to ten minutes and recurring at intervals of a few hours. It is worthy of note that the diagnosis of paresis was made without the presence of delusions of wealth and well-being and was based upon the physical signs, convulsions and facial appearance.

Although it is an accepted fact that there are cases of paresis in which delusions of grandeur are absent either at the beginning or later in the disease, nevertheless, I think such cases are sufficiently rare, to cause one to hesitate before committing himself to a diagnosis of paralytic dementia. It hardly needs to be said that these cases were diagnosed by men accustomed to seeing yearly



CASE I.—FIG. 1. Diagrammatic representation of the brain, showing relative position of gummata in anterior lobe of right hemisphere which are located in white matter and do not appear on inner surface of the brain, as shown in the diagram.

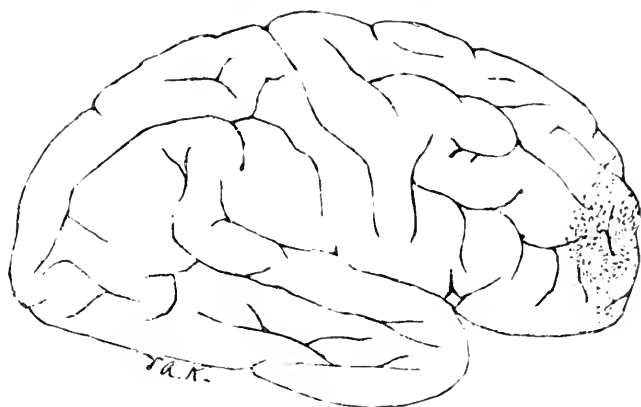
many cases of general paresis in its different stages and various manifestations, and the mistake in diagnosis was undoubtedly due to the non-recognition of the relation of the symptoms of grandeur to that of general paresis.

An autopsy on the brain revealed three large syphilitic gummata in the centre of the right anterior cerebral lobe, which, instead of beginning on the surface of the brain or in the membranes, as is commonly the case, had their seat in the white substance, and by a gradually extending inflammatory process involved to a slight extent the pia and dura. These gummatous deposits, averaging one half inch to one inch in diameter, had begun to soften and break down centrally. The pia showed chronic inflammation with some thickening which was especially prominent along the course of the vessels



about the Rolandic fissure. Carefully prepared sections by the fresh method were examined by the microscope. There were but few changes in the walls of the capillaries. In places they were surrounded by infiltrated leucocytes, but these did not extend to the adjacent brain cells. No erosion of the dendrons or turbidity of cell element were noticeable. In fact, the motor and the giant ganglion cells of the third and fourth layers respectively, seemed to have suffered, but little, if at all.

This case lends additional weight to the fact that syphilis is present in many cases of insanity where a history is or cannot be obtained to that effect, and again where no signs are apparently present indicative of its



CASE I —FIG. 2. The dotting in diagram shows the area of brain surface involved by the extension of the tumors to the cortex. The pia and dura were adherent over same area.

ravages. While it may be argued with considerable plausibility that the presence of the syphilitic gummata in this case, does not militate against the concomitance of paresis, yet I am inclined to think, that all the symptoms given may be accounted for by the presence of the gummata alone. But if we are to tabulate syphilis of the brain, in its many forms, in the same category and speak of it as identical with paresis, as a few writers on the subject are doing at the present day, then, the two diseases might be associated in this case. As this view of the subject does not seem to be fully borne out by clinical facts and pathological observations, I would call the case one of syphilis and leave the burden of proof to those desirous of correlating the two.

The duration of illness in this case was about five

years. It is quite improbable that at first he was suffering from the tumors of the brain as their duration is generally about two to three years. Probably the mental condition on admission could be accounted for by the syphilitic meningitis which preceded and accompanied the development of the gummata.

The clinical history of Case 2, multiple sarcoma of the dura, was as follows: J. B., admitted to the asylum June 10, 1893; 34 years old; German; married and a



FIG. 3. Tumors and outer aspect of calvarium.

moderate drinker all his life. There were no hereditary tendencies found in the family. The patient had been sick for seven months previous to admission and had been out of employment for two or three months prior to the illness, and, in consequence, had attempted suicide by use of the razor. He was confused in conversation and irrational in his behavior. Throughout his entire illness, he was intensely depressed and when questioned, replied in monosyllables. His articulation was slow and hesitating, and at times he broke down entirely, becoming emotional and unable to answer questions. There was muscular tremor in tongue, facial muscles and

hands. The knee-jerk was increased equally on both sides to an arc of about sixty-five degrees. There was no loss of power in the upper extremities, but the fingers of both hands were tremulous. The pupils were unequal in size (record does not say which was the larger), but were regular in outline and responded both to light and accommodation, the response being slow but decided. He failed rapidly in physical and mental health. The circulation was especially enfeebled. The pulse was



FIG. 1. Inner aspect of calvarium, showing the destruction of bone by the tumors. X, cortical area directly under the largest tumor.

slow and almost imperceptible in the left radial artery, while the right was much stronger. No satisfactory explanation has been offered for this inequality of arterial tension, nor did autopsy throw any light upon the subject, although diligently searched for.

Patient complained of frequent headaches, or rather "heaviness of the head," with attacks of vertigo. About once in three weeks, he had mild convulsive seizures which lasted intermittingly for half an hour. These were followed by yet more depression, the patient refusing absolutely to move. These seizures were not epilep-

tiform as they had no aura, biting of the tongue, frothing at the mouth or loss of consciousness. Neither were they apoplectiform as consciousness was not lost, the face did not become congested nor did paralysis occur during or after these attacks. On account of the retention of consciousness, the character of the muscular movement in the seizures, and the recurrence of the attacks in such short periods of time, it was thought that these various phenomena pointed to parietic convulsions and diagnosis of paresis was held until autopsy. The urine was carefully examined during one of these attacks, but with negative results. (Sp. gr. 1.004). These convulsions were general and bilateral and gradually increased in frequency and severity until the 22d of last February, when he died in one of the seizures. The duration of the disease was about one year and six months.

Autopsy revealed multiple sarcomata of the dura, associated with calcific and bony tissue, the presence of which in the sarcoma might render the condition more accurately described if they were called osteo-sarcoma, but as the association of calcification and bony tissue was present in only one of the tumors, it was thought that the diagnosis given was more correct. The presence of the bony tissue seemed to be due to gradual extension by pressure and inflammatory process of the tumor into the wall of the calvarium. There were four of these tumors situated as follows: The largest, which contained the bony and calcific tissue was about the size of a large hen's egg, and was located on the right side, just over the descending parietal region. It was firmly attached to the dura and had probably slowly increased in size, giving rise to an increasing pressure over the motor region, thus accounting for the seizures. Why it did not in the beginning give rise to local spasm of muscle as pressure over this area should do, I cannot say. This tumor had made its way entirely through the calvarium, but did not bulge beyond the cranial surface. It had been so firmly infiltrated that there was no perceptible difference to the touch in moving the finger from the tumor to the adjoining calvarium. These two facts must have accounted in a measure for the non-recognition of the sarcoma before death.

The other three tumors were small, ranging in size from a pea to a hickory nut, being soft and very vascular. They were grouped closely together over the

superior parietal region. But on removal of the calvarium, only one of these three remained attached to the dura, the other two coming away with the forcible removal of the skull cap. The position of the largest tumor was found to be directly over and surrounding the middle meningeal artery. The other three were on the posterior branch of the same artery which gave rise to the idea that the primary cell element of the latter group had been swept there by the circulation from the large sacoma. None of the tumors in this case involved the pia or even the inner surface of the dura. The large one had exerted considerable pressure on the underlying brain tissue, sufficient to flatten the convolutions in that region and give rise to the motor symptoms of convulsions. Microscopical examination by the fresh method of the brain cortex in this region, revealed but very little. The blood vessels showed the most change: their coats were infiltrated by some new cell growth, but the growth did not extend beyond the adventitia. A few of the ganglion cells of the third and fourth layers were thought to show vacuolation, but as only a few sections presented this appearance, it was impossible to say whether it was due to the disease or an artefact. In the accompanying photographs of this case, which were kindly furnished me by Dr. Pettit, the tumors, inner and outer aspect of the calvarium are shown. Fig. 3 shows the small tumors near the longitudinal fissure over the superior parietal lobule. On microscopical examination, Dr. Van Gieson concurred with the above statements of the pathological conditions present. It is a matter of regret that a more careful and thorough observation was not directed to these cases so that the records might have been more complete and definite.

However, it seems reasonable that these two cases of cerebral tumors mistaken for paresis, although insufficient in number to give them any great statistical value, should nevertheless deter us from making the diagnosis of the latter disease in the absence of many of the important symptoms, such as grandiose ideas and feelings of well-being.

Especially should this suggestion have weight as there has been a constantly growing tendency among alienists and neurologists, to diagnose paresis in many cases of mental diseases in which the evidence only a few years ago was thought to be wholly insufficient.

## A CASE OF PSEUDO-MUSCULAR HYPERTROPHY.<sup>1</sup>

By AUGUSTUS A. ESHNER, M.D.,

Adjunct Professor of Clinical Medicine in the Philadelphia Polyclinic, Registrar in the Neurological Department of the Philadelphia Hospital, etc.

CASES of pseudo-muscular hypertrophy are, on the one hand, not so rare as to be considered curiosities, nor, on the other hand, so common as to be without interest. Strictly speaking, the affection does not fall within the category of nervous diseases, being, so far as is known, a disorder of the muscles, pure and simple. In all likelihood, its occurrence depends upon some embryonic defect, some imperfection in the mesoblastic layer of the developing ovum, from which the muscular and fibrous tissues are derived. The designation pseudo-hypertrophic paralysis or pseudo-muscular paralysis is objectionable, inasmuch as the loss of motor power is but an incident, a secondary phenomenon in the symptom-complex.

This case is presented upon the suggestion of Dr. Weir Mitchell, in whose service it occurred, and to whom I owe the privilege of making this report.

J. R., ten and-one-half years old, presented himself at the Infirmary for Nervous Diseases on January 4, 1895. He was born at term, without complication, and during infancy had been fed at the breast. At the age of nine months he had whooping-cough, and shortly afterward chicken pox. He learned to speak at the usual time, and his teeth appeared normally, though with some difficulty of minor character. He has never had a convulsion. No abnormality was noted until the child should have walked but failed to do so. For a time, at the age of about two years, he was able to crawl and creep, but he never walked unaided. He has tried crutches and braces, but without noteworthy assistance. For five years he has been using a tricycle. He is unable to start himself, but once set going he can continue

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<sup>1</sup>Read at the meeting of the Philadelphia Neurological Society March, 25, 1895.

the propulsion. He has been at school for but a few months, and has received but little education. He appears, however, bright and intelligent. The muscular inability has grown steadily and progressively worse.



The boy is entirely unable to stand unsupported. Even when seated, the lumbar spine yields in a forward direction, leaving a marked concavity behind and the sacrum prominent. The legs and feet can be variously moved, though somewhat feebly; the thighs, also, but in somewhat less degree. Flexion of the thighs is particularly weak. The legs and feet are intensely cyanotic; in some places bluish, in some deep red, in some pink. They are also cold, and their cutaneous covering is



rough. The calves of the legs and the buttocks are distinctly full and firm, but not indurated. The musculature of the remainder of the body is rather spare. The child is unable to flex the trunk forward, and rotates the spine little and with difficulty. The movements of the upper extremities are well performed, although the muscles are small. The knee-jerks cannot be elicited, nor can the muscle jerks in the lower extremities. The various reactions in the upper extremities are preserved. The several cutaneous reflexes are maintained. Cutaneous sensibility is likewise preserved.<sup>2</sup> The head ap-

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<sup>2</sup> A separate study of the reflexes and of sensibility was made by Dr. F. S. Pearce, whose notes are appended: Superficial reflexes—Infraorbital present; pupils respond normally; epigastric, abdominal and



pears large; the ears are large; the complexion is pale. The appetite is excessive, and the child overeats at times. The bowels are regular, and the sphincters are continent. The hand-grasp is exceedingly feeble, but it seems alike on both sides, although the parents express the opinion that the right side is the weaker. The disposition is good and the child is bright and cheerful. The rhythm of the heart and the character of its sounds suggest mitral obstruction, but no murmur can be detected. Dr. A. G. Thomson reports that the ocular media are clear; that the fundus and optic disc are normal, although pulsation is evident in the right eye; and that the muscular balance is normal. Nearly three years ago the feet were operated upon, but a tendency to valgus remains. In the family history the only noteworthy points are that a baby-brother died soon after birth in consequence of injuries received from dystocia due to unusual size; that a sister died at the age of four years, having had spinal curvature and finally meningitis; and that another sister died at the age of nine months during dentition. Three other brothers are well and present no obvious abnormality.

For the photographs I am indebted to Dr. H. P. Boyer.

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cremasteric especially active; gluteal less so; erector spinæ cutaneous reflex good. Deep reflexes—Knee jerks absent, not reinforceable; no ankle-clonus; no Achilles' tendon response; elbow-jerk preserved, but faint; no contra-lateral jerk; jaw-jerk present. There is fibrillary contraction generally in the chest-muscles and girdle-muscles on tapping; also in the arms and forearms, but wanting in the legs. The feet are cold and covered with cold perspiration. From the buttocks down there is marked pseudo-muscular hypertrophy. Sensation everywhere is preserved to touch, pain and temperature, but is the more acute in the upper extremities. There is undue mobility at many of the joints, including those of the spinal column. The child assumes an attitude of lordosis in the endeavor to maintain a position of equilibrium in sitting. There is a marked increase of the connective tissue over the lumbar vertebrae.

A study of the electric reactions was made by Dr. J. H. W. Rhein, who found only quantitative changes, particularly marked in the lower extremities. The left biceps and rectus femoris and the right rectus, as well as the triceps of the left arm, failed to yield any reaction to the strongest current that could be employed.

## A CASE EXHIBITING SYMPTOMS OF FACIAL HEMIATROPHY AND JACKSONIAN SENSORY EPILEPSY.\*

By THEODORE DILLER, M.D.,

Visiting Physician to St. Francis Hospital, Pittsburg, Pa.

FOR the opportunity to study this case and present the patient here to-night I am indebted to my friend, Dr. R. W. Stewart.

For want of a better name, I have called the parasthetic phenomena he exhibits Jacksonian sensory epilepsy.

M.C., aged 29, moulder; married; has two children; denies syphilis. Five years ago he began to drink heavily, and continued the habit until two years ago, since which time he has been temperate. His family history is negative.

Ten years ago he noticed that the right anterior part of the scalp was dry, and that he perspired less in this than in other regions of the head. This condition continued unchanged up to the time of injury to be described now.

Four years ago he was kicked by a mule on the right forehead, just over the region which is now most atrophic. He was unconscious for about one minute. The skin was not broken, but the eye and forehead became blue and very swollen, and he was compelled to discontinue work for about a week, when he resumed his usual occupation.

Six months after this injury he had his hair cut very close, and, for the first time, he noticed a bare streak on the scalp, parallel with, and just to the right of the median line. It extended to the line of the hairy scalp on the forehead, and its area could just about be covered by the first two phalanges of the little finger; almost as

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\*This paper was read and the patient presented to the Philadelphia Neurological Society at the meeting held March 25, 1895.

large as you now see it. Over this area, and over a strip of skin, representing a continuation of this region, extending down to the right side of the nose, he noticed that he perspired much less than he did in other parts of the head and face.

Two and a half years ago, about the time of his first epileptoid attack, the patient states that the skin over the anterior two-thirds of the right half of the scalp began to waste, and has continued to do so up to the present time. About the same time he received a blow from an iron rod (not very severe) over the lower part of the right calf, just above the tendo Achillis. A few minutes later his first "spell" occurred in the following manner: He experienced a feeling of numbness and tingling in the left toes which he likens unto a feeling of "pins and needles." This paresthesia slowly ascended the leg, thigh and trunk, went down the left arm to the fingers, then up the left side of the neck and face, involved the left side of the tongue. The attack terminated in pain on the right side of the head, its intensity being greatest in the atrophic regions. When the paresthesia had reached the middle of the trunk in its upward march, it began to fade from the toes, and precisely as it had come it passed away. An absolute loss of muscular power passed over and faded from the left side, just as the paresthesia had, save that, in time, it was a little later. The attack proper lasted from twenty to thirty minutes, but the right-sided head pains continued for an hour or two. He was entirely conscious throughout the attack.

Subsequent attacks have been similar in character except that nearly half of them began in the left fingers. Those which began in this way marched down the left leg to the toes, and then up the neck and face, terminating in head pains, as in the first attack. In a few instances the paresthesia marched down the arm after having ascended it. Two attacks began in the left lip, marched down the arm and leg and returned up the leg and then pursued the usual course. Sometimes the head pains, in which attacks always terminate, last nearly a whole day. This pain is always strictly confined to the right side and is chiefly located in the atrophic regions. The pain varies greatly and is always proportionate to the severity of his attacks. There is considerable difference in the severity of his attacks, and they last from twenty to forty-five minutes. In every attack, no matter

how it begins, the entire left side of the body has been involved. Since the first attack he has averaged about one a month, although they come at very irregular intervals, *e. g.*, six or seven months intervened between the first and second attacks, and two attacks have occurred within three days.

One year ago he was seized with a general motor convulsion, lasting twenty minutes, during which blood tinged foam was noticed about his mouth, and after which he slept. He was, however, conscious during the onset of this attack, and states that it began with the ordinary sensory manifestations in the leg, which were more severe than usual. The last thing he noticed before unconsciousness ensued was that his leg was trembling violently (convulsive movements). (?)

Until the occurrence of this attack, he felt well in the interval between his parasthetic seizures. Since then he has been subject to attacks of dizziness and various ill-defined subjective sensations in the head, and also to certain vague fears. During the past year he has not felt able to work on this account. Since this general convulsion, his sexual appetite has declined until now it is almost absent.

Examination March 3, 1895.—The skin of the right forehead and anterior third of the right scalp is decidedly wasted. There is also slight wasting of the skin over the right side of the nose. This atrophy extends to the median line, but is strictly confined to the right side. The atrophy of the scalp is not even. There are spots where wasting is much more marked than at others. Besides the bald spot already described, there is another one which could be covered with a silver quarter dollar, situated a little back and external to this one. The margin of hairy scalp is a little further back and external to this one. The margin of hairy scalp is a little further back on the right than on the left side. The hair all over the atrophic region of the scalp is less abundant and somewhat coarser than on the other side. Sensation is present over this atrophic region. The orbicularis palpebrarum and the occipito-frontalis muscles are active—apparently as strong as on the affected side.

With the dynamometer he registers sixty-five with the right and sixty-two with the left hand. No disorder of sensation of any sort is present on any part of the body; no atrophy; gait natural; knee-jerks sluggish.



Dr. Frank Edsall, who has examined his eyes, reports as follows: Vision O. D. 20 'xl with + 0.50 D. Sph. 0.50 Deyl. ax 180 = 20 'xxx O. S. 20 'xxv; with + 0.25 Deyl. ax 90 v = 20 'xx. No mydriasis; no muscular abnormality detectable; no limitation of visual field for white in either eye; field for color not tested; small conus of pigment atrophy at the lower temporal quadrant of the right disc; entire disc slightly hazy and indistinct; vessels tortuous, temporal half of the disc a little paler than normal. In the left eye the disc is also a little paler in the temporal half, slight cupping of disc small spot of pigment atrophy at the temporal side of the disc. Disc slightly oval in vertical axis.

#### REMARKS.

The conditions present in the tissues of the right side

of the head leads me to diagnose the case as one of facial hemiatrophy. Apparently the mule kick he received four years ago hastened, if it did not start, the atrophic process. It must be borne in mind that the patient states that ten years ago he noticed the right side of his hairy scalp was crisp and dry; so for six years before the injury some malnutrition existed. When we "remember the fact that the nutrition of all parts except the muscles seems to depend on the posterior root fibres, to which the fifth nerve chiefly belongs, and that the influence of chronic lesions is to cause a slow wasting, distinct from the acute disturbance produced by irritation," (Gowers, *Disease Nervous System*, Vol. II., p. 870), we might suppose that some chronic affection of the fifth nerve or its nucleus, has been present for ten years in this man, and that it was made more active by the mule kick he received four years ago. In the case of facial hemiatrophy, now classic, in which Mendel performed an autopsy (*Deutscher Med. Ztschr.*, 1888, p. 407), degeneration of the descending root of the fifth nerve and of the cells of the locus ceruleus was found. Assuming that a similar condition is present in this man's fifth nerve and its nucleus, might we not suppose that the degenerated nucleus, acting reflexly as a source of irritation, has provoked these periodical sensory discharges? I must confess that I have been unable to demonstrate to my own satisfaction any exact mechanism which would on theoretical grounds definitely account for the attacks. The suggestions I have thrown out, even if adopted, would not explain very far and would leave very much unexplained. If we attempt to explain the attacks on the theory of an irritated sensory cortex, the fact that they begin in different parts of the body, arises at once before us as a difficulty in localization. Besides cortical trouble would not explain the atrophic condition present in the right face. (Bremer and Eskridge have recently reported cases showing cortical trophic influence).

These attacks are themselves, it seems to me, analogous to certain types of Jacksonian epilepsy, except that they are sensory instead of motor, and that there is no constant or signal symptom. The essential feature of epilepsy is the more or less sudden and vicarious discharge of morbidly unstable nerve cells. We are too much accustomed to think this discharge must necessarily be motor. Every now and then we see cases like

this, which seem to be clearly chiefly or wholly sensory. I am disposed to think the motor paralysis he describes as following closely after the paresthesia is more apparent than real. He tells me that on one or two occasions he walked some distance after the parasthesia had involved his leg. Because the leg feels "heavy" and very "large," he thinks he cannot use it. Indeed, it may be fairly doubted whether there is any real paralysis.

The morbid process, wherever or whatever it is, is likely extending. The general convulsions from which he suffered one year ago, the vertigo, the restlessness and uneasiness, the loss of sexual appetite—indicate this. I have as yet instituted no treatment, but have been holding under advisement the propriety of having excised the various branches of the fifth nerve, as recommended by one of the members of this society (Dr. Dercum) in a paper read before the society a few years ago, or of having the Gasserian ganglion itself excised, which, in operation as shown by Stewart, Keen, Rose, Hartly and others, can be done without any very great risk. Dr. Dercum's recommendation (*JOURNAL OF NERVOUS AND MENTAL DISEASE*, Volume XVII. p. 108) is based on the theory that facial hemiatrophy depends, not so much on loss as on perversion of the trophic influence of the fifth nerve. The question, too, as to whether trephining would be advisable also presents itself. Although this man does not, it seems to me, present symptoms which would justify a diagnosis of brain tumor, nor which would localize a focus of irritation, it is possible that trephining undertaken in an empirical way might result in some benefit. Yet, notwithstanding the absence of clear indications, it is just possible that a brain tumor may be present, for we know that in some instances of intracranial growths many, or nearly all the classical symptoms are absent.

# VALVULAR DISEASE OF THE HEART IN TABES.<sup>1</sup>

By PEARCE BAILEY, M.D.,

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FOR many years an alteration in the heart's action in tabes has been mentioned by neurologists. It was not till 1879, however, that valvular lesions not depending on a rheumatic cause were supposed to coincide with any degree of frequency with the degeneration of the posterior columns of the cord. In this year, Vulpian, in his "Diseases of the Nervous System," calls to notice the frequent association of aortic insufficiency and tabes. Also in a clinic held at the Charité in Paris he states that Charcot has often insisted on the frequent association of these two lesions. In the same year Drs. Berger and Rosenbach<sup>2</sup> reported seven cases of tabes complicated with aortic insufficiency. While five of these cases were women and two men, the authors draw no conclusion as to the common exciting cause, simply stating that insufficiency of the aortic valves is the only valvular lesion in tabes which has come to their observation.

Grasset<sup>3</sup> described twenty-four cases of ataxia with diverse cardiac lesions. He believed that the valvular lesion was directly consequent upon the posterior sclerosis.

Jaubert<sup>4</sup> collected from literature and some cases of his own thirty-seven cases of cardiac lesion coincident with locomotor ataxia. Of these cases, twenty presented valvular lesions. In three, there was dilatation of the aorta; in two, fatty heart and myocarditis. The remaining twelve cases did not come to autopsy, and their clinical symptoms are not given with sufficient de-

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<sup>1</sup> Read before the Neurological Section, N. Y. Acad. Med., March 8, 1895.

<sup>2</sup> *Berl. Klin. Wochenschr.*, 1879, No. 27, p. 462.

<sup>3</sup> *Montpellier Medical*, 1885.

<sup>4</sup> *Thèse de Paris*, 1881.



tail to be accepted as instances of valvular disease. Also with the new light which has been thrown in recent years on peripheral neuritis, it is not certain that all of his twenty-five cases would be diagnosticated to-day as tabes. Balacakis<sup>1</sup> examined fifty-six cases, in which he found three instances of valvular disease.

Angel<sup>2</sup> has reported still another variation. Of twelve cases of tabes, especially examined, in seven there were no murmurs at any time, in three there was a diastolic murmur only after prolonged exertion; and in two, a murmur persisted after some hours of repose; in none of the twelve was there any murmur after a night's rest in bed.

The observations and opinions of later observers differ materially from these quoted above, a fact not without precedent in medical history.

Groedel<sup>3</sup> examined 153 cases, in one of which he found mitral stenosis, two of aortic insufficiency and one of mitral insufficiency consecutive to rheumatism. At a meeting of the Berlin Medical Society, held October 29, 1888, Leyden expressed the opinion that the coincidence of valvular disease with tabes was purely accidental. Gutman said that in a 100 cases of tabes he had seen but three instances of valvular disease; while Oppenheim thought that when the two conditions existed in common they were both dependent on the same cause (syphilis).

At present the prevalent opinion in Germany is that the simultaneous occurrence of these two conditions is purely accidental.

Gowers<sup>4</sup> in England asserts that valvular disease of the heart, especially aortic regurgitation, is the most common of all the complications of tabes. He believes that the cardiac lesion is of syphilitic origin. No less an authority than Marie<sup>5</sup> states that in every four or five cases of tabes sufficiently advanced, there will be at least one more or less marked case of cardiac change.

Believing that an analysis of a certain number of cases chosen at random would give surer results as to the coincidence of these two affections by selecting the individual, than by instances of cardiac change from an

<sup>1</sup> *Thèse de Paris*, 1883.

<sup>2</sup> *Berl. Klin. W'och.*, 1880.

<sup>3</sup> *Deutscher Med. W'och.*, 1888, xiv.

<sup>4</sup> *Diseases of Nervous System*.

<sup>5</sup> *Leçons sur les maux de la moelle épini.*

indefinite number of cases of tabes, I have examined a number of cases as they have presented themselves at the neurological department of the Vanderbilt Clinic, all the cases at the Hospital for Incurables, and have collected from literature the reports of several autopsies. The cases examined at the Clinic were all in the earlier stages and still able to walk. Altogether eleven were thus examined. In none was any murmur present. In three the heart dullness extended to the nipple line. In all, the heart was somewhat rapid, although in only two cases was it over ninety-five. The rapidity of the heart's action may be partly explained by the excitement and fatigue necessarily attendant on a public examination.

The ten cases examined at the Hospital for Incurables were all, with one exception, men past middle life, and all in the later stages of the disease. Most all of these patients are unable to walk, so marked has the ataxia become, and they present all the distressing symptoms and complications of advanced tabes, such as Charcot's joint, optic atrophy, cranial nerve paralysis, bladder paralysis, etc. In several of these there is some cardiac hypertrophy, and also several present some slight acceleration of the pulse. The only case which gives the slightest indication of valvular disease is one who is least characteristic of disease of the posterior columns. He is a man 33 years of age, who gives both a syphilitic and alcoholic history; his pupils, while unequal, respond to light; gait slightly ataxic, and loss of knee-jerk. He also has some pains in the legs. He presents well-marked symptoms of aortic insufficiency, with a loud diastolic murmur, hypertrophied heart and a quick pulse.

The autopsies which I have looked over have been referred to by Möbius in his various reviews of tabes in Schmidt's *Jahrbuch*. The autopsies have been made by such men as Oppenheim, Siemerling, Westphal and Déjérine. They comprise seventeen cases in all, in each of which microscopical examination of the spinal cord had confirmed the anti-mortem diagnosis. The vascular system in all of these was normal with the following exceptions. In four there was atheroma of the aorta, while the valves remained intact. In one the valves at the left side of the heart were atheromatous with dilatation and hypertrophy of the heart; and in one there was slight thickening of the aortic valves at the

line of closure, with myocarditis. These observations of my own with the autopsy reports have led me to believe that while organic cardiac changes occur in a small proportion of cases, their occurrence is not more common in tabes than in any other disease of a senile character. The cardiac changes which have been reported have almost exceptionally had the characteristics of atheromatous degeneration. In very few cases has it been limited to the valves alone, and it seems to have been a matter of chance whether or not atheroma of the aorta extended to the endocardium, and if such extension did occur whether it appeared on the aortic valves alone or whether initially the mitral was involved as well.

**Experimental Infectious Myelitis Produced by the Streptococcus.** Drs. Vidal and F. Bezancon (*La France Méd.*, January 25, 1895), experimented on a large number of rabbits, which they kept under observation for a long time. The streptococci were obtained from eighty-nine sources, and were of different virulence. In 6 per cent. of the number paralytic symptoms were observed, which set in seven days to two months after the inoculation. The onset of the spinal symptoms was generally sudden. In four cases the motor troubles were: Flacid paraplegia, with fever, sometimes diarrhoea, *incontinentia alvi* and respiratory disorders. In one case the paralysis was exceeding, and spread to the right front leg. There was always marked consecutive atrophy. In three cases there were contractions, either general, involving all four extremities, the body and head, or localized in certain parts of the body which gave the animals very grotesque attitudes. The cord, examined in four cases, did not reveal any foci of softening; it appeared normal to the naked eye, but the histological examination showed, in the whole length, diffuse degenerative lesions, affecting the gray and white matter. In the former the large multipolar cells especially, are involved; there was granular degeneration; the prolongations had disappeared. In some cases there was colloid degeneration, and in others degenerated nerve cells with vacuoles were observed. The blood vessels were overfilled with blood, ruptured in several places, and the seat of hemorrhagic infarcts. In the white matter the lesions were principally diffuse, and involved the various systems of fibres; the myeline was granular, difficult to stain, and in some places it looked like large drops of fat. The hypertrophic axis cylinders were varicose and irregular. There were no inflammatory changes in the neuroglia, but it seemed to concur with the degenerative process. The meninges, the roots and ganglions, the peripheral nerves and muscles were unchanged. The enumerated changes are attributed to toxic influences, the nervous centres being impregnated with soluble substances of microbial origin. The degenerative nature of the lesions, without vascular changes, is in favor of this opinion.

MACALESTER.

# A CASE OF TABES ASSOCIATED WITH POST HEMIPLEGIC ATHETOSIS AND UNILAT- ERAL REFLEX IRIDOPLEGIA.<sup>1</sup>

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New York City.

At the last meeting of this society I presented a patient with a symptom complex which was considered to be a great rarity, as I could find but a few such referred to in the literature. The symptoms in this case were not those of any single clinical entity, but presented themselves, to my mind, under the form of athetosis, indicating a lesion in the internal capsule or crus, and locomotor ataxia, pointing to involvement of the posterior roots or columns of the cord. Although no history of syphilis could be elicited and no history of its secondary manifestation could be obtained, and, furthermore, in spite of the fact that treatment against such hypothetical syphilis had given no definite improvement, I assumed that these symptoms were a manifestation of cerebro-spinal syphilis; and, in short, presented the patient as one of cerebro-spinal syphilis of a tabic type associated with athetoid movements. Much to my surprise, three prominent and active members of this society stated that, in their opinion, there was nothing in the case to warrant either the assumption or the diagnosis, and that, in their light, the case was one of multiple sclerosis. It seemed to me then, as it seems to me now, that it is scarcely within the bounds of possibility to confound a disease with such a symptom complex as the one I endeavored to lay before you, with a disease that has such a fairly well marked clinical entity as multiple sclerosis as we understand it and as it is described in the books. I surmised that possibly the opinion of these gentlemen was the result of my hasty and faulty presentation of the subject and lack of opportunity on their

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<sup>1</sup> Read at the March meeting of the New York Neurological Society.

part to properly examine the patient. Having given a considerable time to the study of the case, and feeling tolerably sure that the diagnosis was as given, and particularly as I was supported in this opinion by others who had gone over the case with me, I sent the patient to the offices of the gentlemen mentioned, that they might have opportunity to examine the patient more carefully at their convenience, and substantiate their or my diagnosis. At the suggestion of one of the gentlemen, who considered the case sufficiently interesting to warrant discussion, the patient has been sent meanwhile to many of the active members of the society, and they have either sent their opinions to me direct or are ready to state them this evening.

Since the last meeting I have taken considerable pains to go into the man's past life rather scrutinizingly, with a result that much new evidence has been obtained which, I think you will agree with me, lets in a considerable light on the obscure points in the case, and which likewise tends to uphold the original diagnosis. This is my apology for asking your attention to this matter anew. Before speaking of the new factors in the patient's history I shall ask you to allow me to read the brief notes presented with the patient at the last meeting.

J. J. D., 38 years old, male, married, occupation policeman. Seen, in consultation with Dr. E. T. T. Marsh, November, 1894. Family history good. Denies syphilis and alcoholism, and there are no traces of either to be found. Has never used tobacco or indulged in excesses. The only factor in his previous history that is at all pertinent is the fact that in January, 1886, while attempting to make an arrest, he was thrown against a pillar of the elevated railway, and received a severe cut over the right parietal region. He was not unconscious, but was incapacitated for about three weeks. He does not remember any headache after that or since then. His personal history is that he was all right until about a year ago, when he noticed some involuntary movement of the right hand, particularly on excitement and exertion. Accompanying this was a sense of numbness in the hand and right upper extremity and a loss of dexterity of the fingers of this hand. About this time, either before or after—he says he does not remember—(probably longer), he noticed drooping of the left eyelid: in the beginning this was intermittent, but later he noticed

that he could not open this eye so widely as the other. At this time he had some transient attacks of diplopia. The numbness of the right hand and arm got gradually worse, and it felt heavy and useless. Lately he has had a similar feeling of numbness in the legs, particularly in the right, and a sensation of a firm band fastened around the calf of the right leg. Sexual vigor began to diminish about seven months ago, and gradually reached a state of impotency. He has had no trouble with the sphincters. There has been no headache, no disturbance of speech, no pain, no uncertainty of gait, either by day or night.

Examination shows more or less regular movements of the right upper extremity, particularly manifest in the hand. This movement is neither a clonus nor a tumor, nor is it sufficiently rhythmical, determinate, systematic or uniform to be called strictly athetoid; and there is no tendency to distortion; yet it must be considered an athetosis. It continues during sleep, but to a lesser degree, and it is made worse by stimulation and excitement. The movements are a sort of flexion and extension of the hand on the wrist and irregular movements of the fingers. They cannot be diminished by the strongest movement on the part of the patient. The strength of the right arm is a little impaired, and more in the extensors than in the flexors. There is considerable tactile anæsthesia, but pain and thermal sense remain normal. Muscular sense is impaired, and there is loss of sense of position in this extremity. When a coin, such as a quarter or half dollar piece, is dropped into the palm, he may feel it strike, but he has no idea what it is, nor can he tell when it is removed. Marked ataxia of this extremity, complete loss of dexterity of the fingers, and great overaction on attempts at directed effort. General examination shows: Eyes—Slight nystagmus, narrowing of left eye; slight real ptosis; left eye seems small and retracted; pupil pin head, and does not respond to light, but does react to accommodation. Slight muscular deficiency of superior rectus. Right eye: pupil small, responds to accommodation and to light; no limitation in color or visual field in either. Both optic nerves are pale, the left rather more so than the right. Dr. F. F. D'Oench, who examined them a few days ago, thinks they are possibly in the first stage of atrophy. Vision, R., 20-50; L., 20-40; some hyperopic astigmatism glasses make no appreciable difference. Color perception good; no scotoma.

Slight Romberg. Can hold weight of the body on one leg, but this is more difficult with the right leg than with the left. No twitching of left hand, but fingers have lost their dexterity to some extent. The left upper extremity has a slight feeling of numbness, but objectively sensibility is normal. Both knee jerks completely absent, even with re-enforcement. Extremely slight ataxia of gait. No muscular atrophy. A physician who was consulted a few months ago treated him with increasing doses of mercurials and iodide of potassium, so that when the patient was first seen by me he had a heavy, foul breath, tenderness of the gums, and a patch on the side of the tongue which Dr. Marsh and Dr. Erdmann both considered due to the mercury. These disappeared when the treatment was stopped for a few days.

Although the patient strenuously denied syphilitic infection, and although there was no history or evidences of secondary manifestations, considering the well marked evidences of beginning tabes, the patient was put in the Post Graduate Hospital, and a systematic anti-syphilitic plan of treatment instituted. This consisted of inunctions of unguentum cinereum, beginning with a half drachm and increased to three drachms daily, half to be rubbed in at morning, and half at evening, each rubbing to be upward of forty minutes' duration. After four weeks, potassium iodide in small doses, gradually increased was added until the patient was taking 100 grains daily. This treatment, combined with warm baths, rest in bed, massage and tonic plan of treatment, was kept up for ten weeks, and at the end of that time, the patient had not shown any improvement except the return of sexual potency and improvement in gait. The movements of the hand continued, and the sense of numbness was quite as strong. The treatment was then changed to the chloride of gold and arsenic, the administration of the potassium iodide being kept up, and on this treatment he has continued.

As a good deal of divergent opinion was expressed by the gentlemen who examined the patient concerning the ocular conditions existing, I asked my friend, Dr. Ward A. Holden, to make a careful examination of the eyes. This he has done on two separate occasions, and the results of his examination are embodied in the following report:

"The patient has a complication of ocular anomalies which will be more intelligible if described in detail.

"(a) The left palpebral fissure is narrower than the right, due to a slight and varying ptosis, most marked when the eyes are directed forward, and less marked when they are directed upward.

"(b) In fixation the right eye, which has the poorer vision, turns out and up, and this relation is found in every part of the field of fixation. The lateral deviation is greatest on looking to the left, and the vertical deviation greatest on looking to the right, and the mobility of the left eye upward is greatly limited. The patient had diplopia a year ago, and at times has had it since, though it cannot be produced at present. The original muscular lesion would seem to have been an isolated muscular paralysis of the left superior rectus. There are now secondary contractures and possibly paralysis of other muscles, but such cannot be made out.

"(c) When the patient fixes an object directly before him, both eyes twitch laterally two or three times, and then remain fixed. When he fixes an object far to the right, both eyes continue twitching to the right; and when he looks far to the left, there is a continuous twitching to the left. In looking up or down, a slight vertical twitching is combined with the lateral. This is not true nystagmus, in the sense in which the word is used by the ophthalmologist, but the twitching that may occur with paralysis of the extrinsic muscles of the eye, and with various cerebral and spinal affections.

"(d) The right pupil is of medium size, and reacts sluggishly to light and consensually, and to accommodation. The skin reflex is wanting in both. The left pupil is very small, and does not respond to light directly or consensually, but responds to accommodation. When cocaine is instilled, the left pupil dilates slightly and irregularly, becoming horizontally oval, and even when dilated does not respond to light directly or consensually, but responds readily to accommodation. The myosis seems, therefore, to be due to sympathetic paralysis. The tension is the same in the two eyes. This unilateral reflex irioplegia which has been seen a few times has in the cases reported been associated with mydriasis, and not with myosis, as in this case. The left unilateral reflex iridoplegia indicates a lesion in the centrifugal portion of the reflex arc for the light reaction on the left side.

"(e) The vision is 20-40 R. and 20-30 L., not much improved with glasses. A high degree of corneal astig-



matism in the right. Accommodation normal in each. Fields for white and colors absolutely normal in each. The right disc is pale, within physiological limits; the left disc shows a marked general pallor, with sharp outlines and no change in the vessels. There is probably incipient atrophy of both optic nerves, which has as yet not affected their function.

(signed) "W. A. HOLDEN, M.D.,  
"45 West 39th Street."

This is the substance of the patient's story and the status as obtained by many interviews and examinations. The most persistent and repeated questioning as to possible luetic infection or previous disease did not succeed in changing materially the patient's statements. That something definite might be learned of his previous life and ailments, I went to Dr. J. A. Burke, who had been treating him before Dr. Marsh saw him, and, much to my surprise, he told me that the patient had suffered a number of years before—fifteen or more—an attack of hemiplegia. I requested Dr. Burke to look the matter up, and he has embodied his reply in the following letter:

" . . . I think you are right in your diagnosis. I attended the patient some fourteen or fifteen years ago for what I diagnosed as syphilitic hemiplegia, although I could get no history from him of having had a chancre or secondary symptoms. As near as I can remember, I treated him with mercurial inunctions and the internal administration of iodide of potash, and under that treatment the symptoms of paralysis entirely disappeared. The hemiplegia was complete, even affecting the speech, but after more than two months' treatment he entirely recovered. From that time up to about a year ago I saw him occasionally, but never noticed any evidence of syphilis, and, as far as I know, he did not take any anti-syphilitic medicine. About a year ago he called on me for some slight stomach trouble, and I noticed a little drooping of the left eyelid and a partial paralysis of the left side of the face. I think he was not aware of these symptoms till I called his attention to them. Believing these to be caused by the old trouble, I put him on mercury and iodide of potash. He improved slightly, but not fast enough to suit me, so I gradually increased the dose of potash up to nearly 200 grains three times a day.

Then I eased up on the potash and increased the mercury to salivation, but there was no improvement beyond that which had taken place during the first few weeks. He had evidences of salivation when he left me, and went to his police surgeon. He has always been a temperate man—indulged in no excesses of any kind. I forgot to mention that the weakness and rhythmical movements of the right side came on during the time he was under treatment, and gradually increased until he left my care.

“Very truly yours,

J. A. BURKE, M.D.”

On charging the patient with this omission from his story, he very naively remarked that he was under the impression that he had told to me all about it, and likewise to Dr. Marsh and to Dr. Erdmann, but they are of an entirely different memory.

The mode of onset of the hemiplegic attack was, according to his statement now, as follows:

One evening, after he had served at a bar all day, he noticed that he was particularly nervous and felt badly, and was slow in closing up the store. After that he walked to his lodgings, some blocks away, but he could scarcely drag himself along. When he reached the stoop he sat for a half hour or more, and then succeeded in getting upstairs, although his right leg was almost powerless. He went to bed, and the next morning the leg was somewhat more powerless than on the previous evening, and, in addition, the right hand felt heavy. He arose, however, and stayed about the house during the day. On the following morning the right side was completely paralyzed, which was associated, as Dr. Burke corroborates, with disturbance of speech. He does not think his eye was affected at that time. That this was an attack of cerebral thrombosis there cannot be the shadow of a doubt. The age of the patient, the mode of onset, the duration of the symptoms before the hemiplegia became complete, the absence of loss of consciousness, the comparatively rapid disappearance of the paralysis—in short, the entire history of the case, speak unequivocally in favor of cerebral thrombosis.

If one were to be asked the cause of a cerebral thrombosis occurring in an individual twenty-two years old, who was temperate, who had been singularly free from acute disease, including rheumatism, who gave no

history of injury, or exposure, or illness, it is more than likely that he would answer syphilis.

The patient continued to deny any knowledge of infection. Although it is certainly a fact that persons occasionally suffer from syphilis and are ignorant of it, a careful examination of a patient's previous life from year to year will make this number less. The members of this man's family were, therefore, interviewed successively with the hope of finding one whose memory was more verdant and whose statements were more reliable than the one we had to deal with. Of the family a sister living in the country stated that when the patient was about sixteen years old—that is, four or five years before the hemiplegia, he had a sore on the inside of the lip which lasted for several weeks. The sore, as well as she can remember it, was hard, elevated and eating, and the physician at the dispensary where he was treated dusted a powder on it and gave him medicine internally for quite a time. This is the only point of any interest that could be elicited.

I do not wish to lay undue stress on this labial affection, but to me it is within the bounds of possibility that this may have been a chancre. I do not know of any surgical affection at such an age that would require internal treatment, or that would remain so long. The cerebral thrombosis would then be easily explainable and likewise its prompt response to treatment.

The evolution of symptoms from that time to this seem to me to be as follows: Following the absorption of the clot in the lenticulo-striate artery there remained a considerable thickening of the blood vessels. The regular, active, methodical, non-exhaustive and particularly the temperate life of the patient out of doors served to keep his arteries in a good condition. On reaching an age verging on forty, when the late manifestations of syphilis on highly organized tissues are most commonly seen, the tone, the nutrition, the relation existing between the circulating blood on the one hand and the interior of the blood-vessel on the other, call it what you will, became impaired and the result was farther thickening and surrounding encroachment by vulgar tissue on motorial pathways, which in turn, were sufficient to interrupt uniform conduction: that is, sufficient to produce what may be called an irritation and the result is the choreiform, the athetoid movements which one sees in his right upper extremity.

This same factor, acting upon the posterior roots of the spinal cord and the posterior columns, accounts for the loss of potency, the loss of knee-jerks, the loss of pupillary skin reflex in both pupils, the band sensation around the chest, the feeling of constriction about the calf, the sensory disturbances in part, and the beginning optic atrophy and the uncertainty of gait which the patient had when first seen.

The unilateral reflex iridoplegia is, as Dr. Holden has pointed out in his report, a most uncommon phenomenon; the cases on record being extremely scant. In fact Haddens<sup>2</sup> in a recent article on the subject says that there have been reported to his knowledge only five cases of unilateral reflex iridoplegia. It is worthy of note that of these cases four were in tabics. This I consider an extremely important point.

Some of the gentlemen who have examined the patient and reached a diagnosis of multiple sclerosis, have attached great importance to the twitching movements of the eyeball. This I have been accustomed to think is not a very rare symptom in tabes dorsalis, Charcot's dictum to the contrary notwithstanding. It was first observed by Friedreich who called it nystagmus, and thought it was due to ataxy of the muscles. Berger, who has made one of the most exhaustive contributions to the visual disturbances in tabes dorsalis, says that trembling of the eyes after movement is very frequent in tabes, and was absent but six times in 109 cases. In twenty-six cases of tabes, which I have now under observation and in which this condition has been sought for it has been found no less than five times. I believe that in many cases when a tabic patient is told to move the eyes quickly and forcibly, a slight oscillatory twitching will occur in a large number. This oscillation, it has been observed often, is most frequent in a horizontal direction. Therefore, I think that in this patient instead of militating against the diagnosis of tabes the so called nystagmus would rather tend to favor it.

There are a number of cases on record which are very nearly counterpart of the one before us in which the examination of the nervous system after death has left no doubt regarding the nature of the lesion. Pusinelli has recorded a case which simulates very closely, in its clinical history, this patient. (*Archiv. f. Psychiat.*, xii., 3; p. 766, 1892). His patient became infected with syphilis

in 1860, when he was twenty-five years old; five years later he had an attack of hemiplegia associated with aphasia. In 1879, fourteen years later, there was a clear development of tabes.

In 1883 Damaschino (*Gaz. des Hopitaux*, No. I., 1883) related a case and demonstrated a specimen in which the patient had during life only Westphal's symptom and right-sided hemiplegia. The microscopic examination showed degeneration of the posterior columns of the spinal cord and descending degeneration of the lateral columns, in addition to the lesion in the capsule.

Oppenheim (*Archiv. Psychiatric*, xx., I) cites a case in which Westphal made a diagnosis of tabes in a hemiplegic on the grounds of loss of patellar reflex. Post-mortem examination showed anatomical changes in the brain and degeneration of the posterior columns of the spinal cord. In this same article he relates an interesting case in which there was probably syphilis, and in which the picture of tabes was complicated with paresis of the upper and lower extremity with a great deal of weakness in one-half of the body. Later there was complete hemiplegia and paraplegia. The autopsy showed softening in the corpus striatum, but for the paraparesis there were no anatomical grounds save a degeneration of the posterior columns.

Scattered throughout the literature are the reports of a few cases which have been presented to medical societies in which the symptoms would seem to be more or less similar to the case before you, and in which there would seem to have been no question about the diagnosis.

Lacaze (*Montpellier Medical*, No. 1, 1893) reports a case of tabes with general muscular atrophy and the occurrence of marked involuntary movements of the lower extremities and of the face which continued during sleep. These movements partook of the character of chorea.

Porta, of Milan, has observed athetosis of the right hand in two female tabies (*Bulletino delle Poliambulanze di Milano*).

In 1890 Laquer presented to the Congress of South-west German Neurologists and Alienists two cases of tabes, in which athetoid movements of flexion and extension of the hand, which continued during sleep, were prominent symptoms.

A point that has much interested me is the emphasis that has been put upon the fact that anti-luetic treatment

has not been followed by any remarkable results. It seems to me that this is the rule in tabes and the fact that no great improvement has resulted from treatment, in no way denies the ancient infection or its recent manifestations, such as tabes.

The point has been raised that the absence of knee-jerks in our patient may be congenital. It is well known that in an extremely small proportion of individuals this phenomenon is absent. This point cannot be controverted by the production of positive proofs. It must be allowed, however, that ten years ago this patient had to pass what is generally believed to be a careful examination before a board of physicians in order to get on the police force, and the absence of knee-jerk was not detected by this examination; neither was it remarked by the physician who treated him during the attack of hemiplegia. The return of sexual potency during the last few months, while under treatment, is a corroboration of the well-known fact that one of the earliest beneficial manifestations of treatment in beginning tabes is the stimulation of waning sexual vigor and rather frequently a partial restoration of that function for a longer or shorter time.

The onset of tabes unaccompanied by pain is not common. Yet it has undoubtedly been within the experience of every member of this society to see such cases, and I shall not run the risk of wearying you by citing such cases from my own experience or from the literature.

Discussion as whether this be a case of cerebro-spinal syphilis or tabes dorsalis associated with athetosis is not particularly pertinent or productive of results. Insomuch as it is probable that twenty years ago he had an initial lesion and some years later a cerebral thrombosis, which was more than likely dependent upon a syphilitic affection of the blood vessels, and that now he has the first manifestations of a disease for which syphilis is postulated as causative in from seventy-five to ninety-five per cent of the cases, it might be said, and justly, that his symptoms are dependent upon multiple lesions, primarily syphilitic in origin, and to such an extent the case is one of cerebro-spinal syphilis. The clinical picture presents itself, however, as a combination of two distinct clinical entities, tabes and athetosis, and it is as such that I have taken the liberty of presenting him to you for discussion.

## Periscope.

### PHYSIOLOGICAL.

**Recent Studies on the Physiology of the Cerebellum According to Ferrier.** Rectification and Replies by Luciani. (*Reggio-Emilio*, 1893.) This article was called forth by Ferrier's inaugural address as President of the London Neurological Society. In this address Ferrier, while speaking of his own experimental researches regarding the functions of the cerebellum, discussed and criticised Luciani's views on that subject. Luciani found that Ferrier had made so many inaccurate and erroneous interpretations of his (L.'s) works, that to leave Ferrier's sayings unanswered would be equal to a surrender; that is, to the admission that his (L.'s) painstaking researches, the work of years, were practically without results.

L. begins by discussing the interpretation of the symptoms observed as the immediate effect of cerebellar ablation. He calls these symptoms "irritative," basing this conclusion upon the following facts: They are proportionate to the degree of the operative traumatism, or to the degree of inflammatory process called forth by the operation; they prevail in the muscles of the side, which has been exclusively or predominantly injured. If from the effect of a former extirpation of the vermis, part of the nerve fibres composing the fasciculus peduncle is degenerated, subsequent ablation of a lateral lobe causes only slight symptoms of increased functional excitability.

The irritative symptoms observed by L. in dogs after hemi-extirpation of the cerebellum were disquietude, frequent lamenting howls, pleurothotonus; that is, curvature of the spine, with the concavity towards the operated side, tonic extension of the anterior extremities and a tendency to roll over the longitudinal axis from the operated to the healthy side. After total extirpation of the cerebellum, the symptoms were bilateral, the pleurothotonus being replaced by opisthotonus. In monkeys the tonic extension of the extremities was substituted by tonic flexion.

Ferrier denies the irritative character of these symptoms, and calls them dynamic or inhibitory. L. then criticises F.'s mode of operating; viz., the destruction of nerve centres by canterization. He calls this procedure anti-physiological. He remarks sarcastically that if one intends to bring elements of disorder and confusion into a well arranged, clear and coherent series of experiments, if one proposes not only to destroy a certain centre but to also irritate the adjoining regions, Ferrier's method does excellent service. As an illustration, L. cites three experiments of his own, in one of which canterization of the dura mater over the right side of the cerebellum without injury to the latter caused pleurothotonus on the healthy side, tendency to roll around the longitudinal axis from the healthy to the operated side, and general depression, that is, symptoms which were the opposite of what is observed after hemi-extirpation of the cerebellum.

L. then defends his theory which explains the phenomena observed some time after cerebellar ablations, when the irritative effects of the operation have passed and have been replaced by those which are the consequence of the newly formed cerebellar defect. He classifies these symptoms into three groups; the asthenic, atonic and atactic. He thinks that they are three various external manifestations of one internal process. The objection made by F. that according to clinical experience atasia and abasia of cerebellar origin may exist while in a ly-

ing position yet the patient is able to perform with his legs any movement ordered with great "surety" and "rapidity" (L.'s correction of Ferrier's words is that these movements are performed with "energy" and "vigor") is to L. no proof against the validity of his theory. The above clinical experience only proves (says L.) that the static ataxy observed was neither dependent upon paresis nor paralysis, nor incoördination, but solely the result of asthenia; that is, lessening of energy and vigor of the muscles of the legs, making them unable to support the weight of the whole body, but not to raise and move the legs normally.

L. further calls attention to the manner of swimming by dogs which have been deprived of one-half of their cerebellum. On account of the lessened vigor in the movements of the extremities of the operated side the body is inclined towards the latter so that the operated side is more deeply immersed in the water than the other. For the same reason the animals are not able to swim in a straight line, but are compelled to describe curves with deviation towards the healthy side. Ferrier contends that under the conditions mentioned the reverse would have to take place: viz., the animals would be compelled to describe curves towards the weakened side. To prove Ferrier's error L. justly calls attention to the manner of swimming of the dogs, which consists in downward, inward, backward movements of the paws. The asthenia (continues L.) also shows itself in the station of the operated dogs. They keep the extremities of the operated side in exaggerated, abducted position, thus creating a broader base for the support of the body and diminishing the work of the weakened muscles.

According to L. the second group of disturbances following cerebellar ablations (at the time when the irritative symptoms have disappeared), viz., the "neuro-muscular atony" has nothing directly to do with the tendon reflexes. The author contends that exaggerated tendon reflex would be no proof against an atonic state of the muscles in question; as a rule some time after the operation the tendon reflexes become indeed somewhat exaggerated.

One manner of manifestation of the neuro-muscular astasia is the exaggerated manner in which the limbs are raised and put down in walking. L. has no objection to make against Herbert Spencer's (cited by Ferrier and Ross) hypothesis concerning the function of the cerebellum. The result of his researches harmonizes with the theory that "the cerebellum regulates the continuous and tonic muscular contractions." L. makes a distinction between this continuous functional influence of the cerebellum.

The author feels complimented that his researches have made such a deep impression on Ferrier's mind, and particularly that he now no more speaks of the cerebellum as of an organ of equilibrium, nor as of an accumulation of unconscious centres of reflex adaptation. If Ferrier had not felt too sorry for being compelled to give up his previous ingenious hypothesis concerning the physiology of the cerebellum he would now be one of the strongest, and certainly most competent, supporters of his (L.'s) doctrines.

In conclusion, the author calls attention to some of the inexactitudes in Ferrier's last publication. As an example, he mentions that according to the corresponding photographs, two-thirds of the cerebellum had remained intact in a case which Ferrier and Turner described as complete extirpation of the cerebellum.

ONUF.

*Is there Hysterical Nystagmus?* Sabrazes, (*Semaine Med.*, Sept. 26, 1894).

Such is the question which the author answers in the affirmative. After discussing the various affections in which nystagmus may be an important symptom, and after quoting the dictum of Charcot, that nystagmus is never found in hysteria, the author relates a case, which is



evidently hysterical, in which nystagmus was well marked, and not dependent upon any ocular defect. To prove its hysterical basis, the author cites the fact that it disappears under hypnotic suggestion, and that it was accompanied by retraction of the visual fields. J. C.

**Crossing of the Respiratory Paths in the Cord.**—Porter (*Centralb. für Physiol.*, viii. 7, 1894).

The author concludes: 1. That the respiratory paths ascend in the lateral columns of the cervical cord. 2. That stimulation from the medullary centres can excite, unless dyspnoea prevents, the phrenic centre of the other side. 4. This discussion occurs at the level of the phrenic centre. 4. Such a discussion does not occur in the cord between the phrenic centre and calamus scriptorius. J. C.

**The Trophic Properties of Nerves.**—Gaule, (*Berlin. Klin. Wochenschr.*, Nos. 44 and 45, 1893.) The contention of the author is that trophic properties are not dependent upon special trophic nerves, but that such properties are furnished nerves by their ganglionic cells. To the objection of Eckard, that if such be a fact then we should not get atrophy by section of the trigeminal centralwards from the Gasserian ganglion, which the latter has frequently produced, Gaule replies that he has repeated the experiment of Eckard and is convinced that the nerve contains as far as this point: *i. e.*, a point considerable distance from the pons a great number of ganglionic cells.

Injury of the third, fourth, fifth and sixth spinal ganglion in the rabbit is followed by trophic troubles in the skin, the glands and muscles, not alone on the same side as the injury, but on the other side as well; and not alone in the territory of the corresponding ganglion, but also in the territory of other nerves. When the ganglion has previously been denuded, irritation and lesions are without result. The alteration which it produces in the ganglionic cells effects results by being carried to the spinal cord through the posterior roots. In order to obtain trophic lesions in the rabbit which shall be confined to the same muscles, the author experimented on the inferior cervical ganglion of the sympathetic. The changes that followed were in every instance confined to the same portion of the brachial biceps, and the *psoas iliacus* and showed identical characteristics. As in some of the cases, the affected muscles were on the same side as the experiment was performed on, sometimes on the opposite side, and sometimes the muscles of both sides, one is obliged to admit that the transmission was effected through the spinal cord by the aid of the communicating branches of the sympathetic with the spinal nerves.

Gaule was able to follow the evolution of the alterations which went on in the midst of a muscle by the aid of his discovery of a nerve which stopped at a place adjoining the spinal ganglion. The irritation of this nerve-branch made the ganglion insensible to excitation, in spite of the fact that that section of the nerve caused exaggerated ganglionic sensibility. A few moments after excitation of the ganglion the muscle lost at one point of its surface its soft silken feeling. Then appeared a liquid at this point, and the surface of the muscle began to show lowered nutrition and to become rugous.

This depression involved the entire muscle, both on its external and internal surface. In the median portion it extended as far as the tendon, from which it was separated by a languette. In from five to ten minutes an ulceration was produced which was characterized by its very red color and with elevated borders. Often the ulceration was so extensive that it sufficed to cut the muscle in two. Not infrequently a considerable hæmorrhage followed or attended such ulceration, and this hæmorrhage penetrated into the neighboring muscular fibres and the connective tissues. The succession of phenomena in such conditions is as follows: The first alteration is a softening or liquefaction of the muscular fibres at the determinate point. The fibres do not break

down unless there is a contraction or distension of the muscles, but rather because of the normal state of tension of the muscles which the altered fibres are no longer capable of resisting. Once the fibres break, their loose ends retract themselves, thus forming a nervous thickening around the ulceration. From the very beginning one is able to distinguish microscopically in the muscular fibres cavities which become filled with liquid in spite of the fact that the rest of the substance continues red as it approaches the interior of the sarcoloma. J. C.

#### PATHOLOGICAL.

**Examination of the Blood in Myxœdema.** Drs. Libreton and Vaquez (*La France Méd.*, January 18, 1895) presented a patient with congenital myxœdema, treated with thyroid. The diameter of the red globules before the treatment began was 3 M 13; afterwards it was 7 M 5. At the same time the appearance of nucleated red globules was observed, which disappeared under treatment. It would appear as though the persistence of the foetal state of the blood coincided with the tardy development of the body. Kraepelin observed the same modification of the blood two years ago. MACALESTER.

**Scleroderma in Plaques.** Dr. Hallopeau (*La France Méd.*, January 18, 1895) presented a case of *sclerodermie en bande*, limited to the area of the internal cutaneous brachial nerve. The disorder began as a spot in the middle of the forearm, and only after several months extended the whole length of the upper extremity. If, as is probable, the affection is a tropho-neurosis, the different fibres of the nerve were involved successively. The onset and course were marked by paræsthesia and lancinating, cutting pains. In the sclerosed parts there was a diminution of sensibility to touch and pain, which is generally not the case. MACALESTER.

**The Influence of the Thyroid on the Amount of Oxyhæmoglobin in the Blood.** Dr. Paul Masion (*Bull. de l'Acad. roy. de Méd. de Belgique*, iv. ser., vol. ix., No. 1) in an elaborate experimental *memoire*, comes to the following conclusions: 1. The relative amount of oxyhæmoglobin is diminished after thyroidectomy. 2. The chart of the diminution follows, markedly, the course of the injuries which follow thyroidectomy. 3. During the epileptiform seizures and nervous accesses there is a mixture of reddened hæmoglobin and oxyhæmoglobin. 4. The diminution of the oxyhæmoglobin does not result from the diminution of the relative number of red globules or from the inanition following the injuries. 5. The diminution is the direct result of the suppression of the functions of the thyroid. MACALESTER.

**Pathological-Anatomical Findings on the Clivus in Insane Patients.**—Dr. R. Schweter. (*Allgemeine Zeitschr. für Psychiatrie und Psychisch-gerichtliche Med.*).

The writer gives a short anatomical description of the clivus. In his researches he finds this structure to vary frequently with regard to its form and direction of surfaces. Especially to be noticed is the frequent and remarkable division of its surface into three parts. In addition, the abnormal line formations, such as ridges running obliquely across its surface, tuberos prominences, and osteophytes, both single and double in number are present. The author finds that it does not possess any distinct relation in regard to its form with the neighboring structures.

Of most importance, for examination, he considers the irregularities on the surface of the clivus, viz.: the echondroses, exostoses and osteophytes. The consistence of the same should not be forgotten.

In a careful examination of the skulls of 316 insane patients with reference to this particular part, he has noted his findings as follows:

One hundred and sixty-five were males; 151 were females. One

case, the history of which has been carefully recorded, showing symptoms referable to the delicate structures in the neighborhood of the clivus, there was shown at the autopsy to be present besides an endocarditic adhesion with the heart, atheromatous condition of the arteries, coronary and basilar, suppurating areas in various parts of the body, atrophic frontal lobes, an exostosis upon the clivus. This exostosis proved to be the upper portion of a strongly developed and high-reaching odontoid process of the axis, which undoubtedly became separated from it through the suppuration which was going on in its neighborhood.

He found this condition present only twice amongst his 316 cases.

Osteophytes were present 49 times. In the majority of cases they were single. In form, sharp and pointed.

In two females the second osteophyte, where they were double, was replaced by a rough bony ridge.

In one male and two females the bony ridge alone was present.

In a great number of cases he found tuberos bony prominences.

Noticeable tuberos elevations were frequently seen on the edges of the clivus in the neighborhood of the dorsum.

Often there was noticed a groove, which corresponded with the position of the basilar artery.

To be mentioned also was the diminished consistency and the interruption in the continuity of the surface of the clivus in a few of the cases.

With regard to the ætiology, there was no marked presence of scrofula, tuberculosis, or syphilis.

With regard to psychical disturbances, the majority were male paralytic cases.

The symptoms which might have been due to these abnormal bone formations on the clivus cannot be established as a fact. The space is a very narrow one, and although pressure in this region occasioned by such growths may compress the delicate nervous structures in the neighborhood (the medulla-vagus-hypoglossal-facial nerves and the blood and lymph channels), the latter bringing about nutritional changes, nevertheless, the author is inclined to believe that a sort of compensation in the arrangement of these parts may take place. That is, they alter their position in such a manner as to accommodate themselves to this space which has been encroached upon by these abnormal growths, and thus avoid compression. They may either be drawn higher up in the cranial cavity or pressed toward one side.

WIENER.

#### CLINICAL.

**Treatment of Epilepsy, With Especial Reference to the Use of Opium.**—Joseph Collins. (*Medical Record*, September 22, 1894).

About a year ago, Professor Flechsig, of Leipzig, published a short article on a new method of treating epilepsy, which, in his hands, had given most gratifying results. It consists in administering opium in the shape of the extract or pill in large doses for a period of six weeks. The dose of opium in the beginning is from one-half to one grain, and this is gradually increased until the patient is taking about fifteen grains per day, in doses of from one to four grains. The maximum dose is reached by the end of the first week. At the end of six weeks the opium is stopped suddenly, and for it bromide of potassium or sodium, in doses of one-half drachm four times daily, is substituted. After these large doses of bromide have been kept up for some time, the dose is gradually decreased until the patient is taking less than two scruples per day. The sudden cessation of administering the opium and the exhibition of the bromide are quite essential.

Being cognizant of the fact that opium in small doses, when combined with bromides, is frequently of great service in lessening the severity of attacks of epilepsy, the plan of treatment suggested by Flechsig appealed to the writer, and he determined to give it a thorough trial in a large number of cases selected from private, hospital, and dispensary practice, and examine the results of such treatment at the end of one year. About fifty persons have been subjected to this plan of treatment. A few cases he details.

The twenty cases considered in his first table he had under almost daily observation, and the conclusions reached regarding this plan of treatment are based largely on the facts therein set forth. The sixteen cases considered in the second table he did not have under such close surveillance. They were very old subjects, and his principal desire in subjecting them to the treatment was to determine whether the results would militate against or corroborate the results obtained in the first twenty cases. In a general way it may be said that the results were not so satisfactory.

A study of the cases shows that all of them, with one exception, were benefited. It further shows that in two cases the fits have not returned. The time elapsed in these cases is, however, not of sufficient length to refer to them in any way as cured; and, although the attacks have ceased, the author does not consider them cured. In several of the cases the character of the attack after the relapse was frequently of a different nature than that before the attack,—that is, the family of the patient or the hospital nurse, as the case might be, would describe them as "mere fainting spells?" and make light of them. He observed, however, as time went on, that these "spells" gradually became more severe as the number of attacks increased after the relapse. Another fact which is pointed out is, that in these cases, almost without exception, the maximum dose of opium was reached and preserved in with comparatively slight trouble. Some of the patients who were cognizant of what they were taking complained rather bitterly and rebelled, but particular attention to the bowels and a good deal of out-door exercise served to keep them moderately comfortable. Sometimes it was necessary to administer strong coffee to combat the drowsiness, particularly during the first weeks, when the opium was being increased rapidly.

While the patients were taking opium there was no marked change in the frequency of the fits. Some of the patients had a lesser number, particularly towards the latter end of the six weeks' period, while others had them with customary frequency.

Notwithstanding the fact that no sinister results accompanied the administration of opium in such large doses, still, it should be stated that anyone who would apply this plan of treatment must be watchful and scrutinizing, especially during the first weeks, until the patient becomes accustomed to the large doses.

The most satisfactory results were obtained in very chronic epileptics, and particularly those who were not responsive to large quantities of bromide. In epilepsy dependant on or associated with gross organic lesion of the brain the treatment seemed to give better results than in pure idiopathic epilepsy. By gross organic lesion is meant epilepsy associated with defective development.

The writer sums up his conviction as follows:

1. The plan suggested by Flechsig is not a specific in the treatment of epilepsy.
2. In almost every case in which this plan of treatment has been tried there has been a cessation of the fits for a greater or lesser time.
3. A relapse generally occurs in a period varying from a few weeks to a few months.

The frequency of fits after the exhibition of opium is, for the first year at least, lessened more than one-half.

5. The attacks occurring after the relapse are much less severe in character than those that the patient has been accustomed to having.

6. This plan of treatment is particularly valuable in ancient and intractable cases.

7. In recent cases of idiopathic epilepsy it cannot be recommended.

8. The opium plan of treatment is an important adjuvant to the bromide plan as ordinarily applied.

9. The opium acts symptomatically, and merely prepares the way for and enhances the activity of the bromide and other therapeutic measures.

10. This plan of treatment permits the use of any other substances which are known to have a beneficial action in epilepsy. J. C.

**Epilepsy Cured by Curare.**—(*Die Therapie der Gegenwart*, Feb. 1895). Dobronrawo reports the case of a sixteen-year-old boy who had been epileptic since infancy, the attacks being preceded by a peculiar feeling of irritation in the right knee. The fits continued to come on with increasing frequency. One day, having been seized with an attack on the street, he was removed to the hospital. Here the spasms continued at intervals of a few minutes. He was given iodide of potassium, bromide of potassium in large doses, antipyrine, salicylate of sodium, and chloral, but all to no purpose. Finally, after twelve hours, D. administered 6 mgrm. of curare hypodermically. Thereupon the frequency of the attacks diminished somewhat, and then subsided the following morning. During the next day he experienced slight weakness in all the muscles, and an indefinite senseless feeling at the level of the right knee. After this, at intervals of fifteen days, five more injections of from 6 to 7½ mgrm. curare were given. No further attacks appeared, and he has now been well for several months. FREEMAN.

**Tetanus Traumaticus Treated With Antitoxin (Tizzoni).**—Von Hacker (*Wien. Klin. Woch.*, No. 25, 1894).

Von Hacker reports a cured case of tetanus treated with antitoxin. The patient was a thirteen-year-old boy, who had cut his thumb. He presented the usual symptoms of the disease. He was narcotized, and the wound which was suppurating, thoroughly scraped. On the same day he received 2.35 g. of antitoxin, and after that doses of 0.6 g., as frequently as the exigencies of the case required. At first the condition of the patient became worse, but soon ameliorated, and on the sixteenth day after the first antitoxin injection, symptoms ceased altogether. The total quantity of antitoxin administered was 4.05 g. MEIROWITZ.

**Four Cases of Trismus and Tetanus Neonatorum Treated With Tizzoni's Antitoxin.**—Escherich (*Wien. Klin. Woch.*, No. 32, 1894).

Of these four cases only one recovered. On the fourth day after birth, the umbilical cord became detached. The wound did not heal promptly and the mother resorted to household remedies. On the eleventh day of its life it was unable to take the breast and was taken to the hospital. It presented the typical picture of tetanus neonatorum with trismus. Evening temperature was 38.8°. The treatment consisted of antitoxin 0.3 g., and sublimate applications to the navel. The following day the tetanic symptoms became worse, and the temperature rose to 40.5°. On the third day, the condition of the child improved, but it was not until the twenty-third day, that the patient was pronounced cured. The following table presents facts pertaining to the four cases

Case.	Body Weight.	Length of Incubation.	Disease.	Injections.	Autopsy.
1.	2,260 g.	2 days.	8-10 days.	2 of 0.015 g.	omphal. sept., peritonitis.
2.	2,780 g.	9 "	12-17 "	2 of 0.25 g.	" " pneumon.
3.	3,418 g.	7 "	11-23 "	3 of 0.3 g.	" " "
4.	3,210 g.	1 "	6-7 "	3 of 0.3 g.	omphal. sept. pneumon.

MEIROWITZ.

**A Severe Case of Tetanus Treated With Subcutaneous Injections of Corrosive Sublimate.**—Celli (*Arch. Ital. di Pediat.*, 1894). Abstract by Toeplitz

In consequence of a neglected wound of the foot, a six-year-old girl developed tetanus and trismus. The wound was thoroughly disinfected, rectal injections of chloral and opium instituted, and subcutaneous injections of corrosive sublimate in 0.0059 doses given. After the second injection, the symptoms diminished in severity and disappeared completely after the ninth injection had been made. The injections were repeated at intervals of twelve hours.

MEIROWITZ.

**Myasthenia Gravis Pseudo-Paralytica.**—Jolly (*Berlin. Klin. Wochenschr.*, January 7, 1895).

Such is the burdensome, but very descriptive name given to a class of cases to which the author called attention by the report of a case about four years ago, and the discussion of which he again opens up by relating the following history: A fourteen-year-old boy had noticed for more than a year before coming under observation that he was able to keep the eyes open only with difficulty and conscious exertion. Sometime later he noticed the most decided myosthenia in the legs, particularly after walking a short distance. Soon after these manifestations, a condition of similar weakness was complained of in the upper extremities and a feeling of complete exhaustion in the muscles of mastication after chewing a few mouthfuls, and in the face, generally, after speaking or reading aloud. Examination showed an absence of any perceptible muscular change; that is, there was neither atrophy nor hypertrophy, the myotatic irritability was preserved and sensation remained unimpaired. Electrical reactions were characteristic of what Jolly calls myasthenic. That is, at first the reactions were quite normal; but continued excitation exhausted the excitability and promptness of response just as did voluntary effort; but a period of rest, the same as after voluntary exertion, was sufficient to restore temporarily the normal excitability.

The author points out that this condition of myasthenia is the opposite condition to that present in Thomsen's disease, but they have it in common that in both the abnormal condition is called forth by voluntary effort and by electrical excitation. He also calls attention to the suggestive fact that two alkaloidal substances, veratrine and proto-veratrine, produce contractions and myasthenia respectively, similar to those of Thomson's disease and myasthenia pseudo-paralytica. In discussing the probable pathology of the affection, the writer remarks that the condition may be the result of divers pathological conditions: disease of the nuclei of certain groups of nerves, in another instance to disease of the pyramidal tracts, the muscles themselves, etc. Therapeutically, all over-exertions, including the application of faradism, are to be carefully avoided. It is not yet safe to say if galvanism, properly applied, is beneficial or detrimental. Tentatively, and under the strictest supervision, such substances as veratrin, whose physiological action is to cause the direct opposite from myasthenia, might be tried.

J. C.

**The Equivalent of Migraine.**—Bary (*Neurolog. Centrblt.*, March 15, 1895). The writer says that although the books speak of the fact that migraine may be represented by pain in other parts of the body he has not been able to find specific examples related in the literature. In this article he records such a case. A fifty-one-year-old woman, of degenerate inheritance, suffered from the ninth year on with headache, which occurred with considerable regularity every three or four weeks, while in the meantime her health remained very good. As directly causative of these attacks were considered, mental excitation, remaining in rooms that were overheated, tobacco smoke, etc.; but the patient has remarked that when she has avoided such exciting influences the attack will come on spontaneously. The severity and duration of an attack is very variable, the lightest last three to four hours, and do not prevent

her from being about; in the severe ones there is great prostration accompanied with severe vomiting and lasting from twelve to eighteen hours. The pain is mostly in the temporal region, and is not preceded by an aura. The ordinary therapeutic measures, including the synthetic analgesics, are of but little service. Four years ago she had an attack of severe epigastric pain which came on unheralded and spontaneously, and unaccompanied by any other manifestations of gastric disturbances. At first it came every day, lasted a few hours and disappeared suddenly. No attributable cause could be found. The migraine never occurred during this time. After a few months the epigastric pains ceased and the migrainous pains recurred, then later yet their disappearance and a reappearance of the epigastric attacks. The writer considers the epigastric pains the equivalent of the migraine.

J. C.

**Cerebral Sclerosis Following Influenza.**—Rendu (*Sem. Med.*, December 26, 1894).

The author relates two cases which he has diagnosed as cerebral sclerosis, in which the appearance of the symptoms were consecutive to an attack of influenza. The first patient, a young man, had a transitory attack of hemiplegia while suffering from the grip, and after recovery from the latter, he remained quite well for about four months, when he began to complain of difficulty in walking and headache, which at first suggested cerebellar disease, but later was attributed to neurasthenia. These symptoms disappeared after a few months duration, and were followed by symptoms suggesting astasia abasia. These in turn lasted a few months and were followed by tremor of the hands and head, exaggeration of the knee-jerks and nystagmus, which the writer thinks points to a disseminated cerebral sclerosis. The second case related by the author occurred in a child who was taken with epileptic convulsions while suffering from post-grippal tonsillitis. The epileptiform attack was followed in turn by a left side hemiplegia, from which the child recovered with the usual remains of muscular atrophy and increased myotatic irritability. The writer believes that the latter case was one of localized encephalitis, which was followed by a localized sclerosis.

J. C.

**Isolated Paræsthesia in the Distribution of the Nervus Cutaneus Femoris Externus.**—Bernhardt (*Neurolog. Centrblt.*, March 15, 1895). The author first refers to a previous article, in which he pointed out the occurrence of a double sided degenerative neuritis of the ulnar nerve following on typhoid fever. In that article he said that he had seen not infrequently cutaneous paræsthesia in the region of ulnar distribution follow typhoid fever. He now says that since that time he has opportunity to verify this peculiar manifestation after typhoid, occurring in the distribution of the external cutaneous nerve of the thigh. He gives the history of a military physician, who had for many years complained of abnormal sensations on the outer side of the left thigh. This did not hinder him very much in ordinary walking, but it was so troublesome in long marches that he sought discharge from service. In his youth the patient had typhoid fever, and since then, the paræsthesia. Several other cases are referred to, and in conclusion the author states that all his cases have been in men of middle life who have complained of abnormal sensations, almost always a feeling of numbness, in the anterior and outer surface of the thighs. Sensations which are increased, especially by long marching and by pressure such as that of a scabbard on the involved region. Spontaneous pain when the patient is quiet, such, for instance, as frequently occurs in neuralgic conditions, is not complained of. Motility in the affected leg remains unaltered. Occasionally an accompanying condition is a slight disturbance of the functions of the bladder and rectum. The objective disturbances of sensibility are very slight, and the author has never found a true anæsthesia. Besides typhoid as a causative condition of this paræsthesia, other factors, such as lead poisoning, application of cold, such as

douches after excessive heating, etc., may be found. The writer has it found that the paresthesia responds to treatment, except to be ameliorated.

J. C.

**Nervous Complications Caused by Muco-Membranous Enteritis.**—(*Gazette des Hôpitaux*, Jan. 29, 1895.) M. Cantru, in "Medecine Moderne," states that in cases of epilepsy, chorea, or other grave nervous manifestations of which the cause remains undetermined especially when no neuropathic heredity exists, we should look to the intestine for the existence of muco-membranous enteritis. If this condition be found, it should be considered the cause rather than the effect of the nervous complication. In such cases it becomes especially important to avoid the employment of the bromides, for when taken by the stomach they not only fail to cure the complications, but rather keep up the malady by their irritating action on the mucous membrane of the intestine. This observation is a very just one, and does not in any way compromise the justifiable reputation of bromide of potassium in the treatment of epilepsy.

FREEMAN.

## PSYCHOLOGICAL.

**The Influence of Alcohol on Sexual Perversions, Epilepsy, and other Psychological Anomalies.**—Aug. Forel (*Deutsche Med. Wochenschr.*, Dec. 27, 1894), calls attention to the fact that the inordinate use of alcoholic beverages not only leads to the development of the ordinary alcoholic psychosis, especially delirium tremens, but it also plays a prominent rôle in psycho-pathology in two different ways: Firstly, the hereditary, pathological predisposition of certain persons is such that they cannot indulge in alcohol moderately, but become dipsomaniacs at once, if they do not abstain all their lives. Secondly, alcoholic intoxication either stimulates or develops directly any latent psycho-pathological germs that might otherwise have remained latent. In the current casuistic of sexual perversions the principal factors enumerated are congenital and acquired dispositions, acquired nervous or mental disorders, and, in some cases, bad habits. There is no mention of alcohol as a causative factor. F. observed cases in which the use—or abuse—of alcohol was a most prominent feature, and enumerates divers illustrative histories. It is an established fact that epileptics stand alcohol very badly, and when intoxicated are especially dangerous. There is also an alcoholic epilepsy, the subjects having fits only when intoxicated. F. also alludes to the "pathological" intoxications, followed by amnesia, and concludes that nearly every psychosis is aggravated by the use of alcohol. Other psychoses, which are either caused or kept up by alcohol are, for instance, alcoholic mania, melancholia, pseudo-paralysis, incurable secondary dementia, acute and chronic hallucinatory folly, etc. F. observed many cases cured by abstinence, and considers indulgence one of the greatest obstacles in the radical cure of morpheo mania. He had always observed that the excitement of the insane in asylums was always greater after entertainments at which wine or beer were served. At Bûrgholzli, of which F. is director, alcoholic beverages have been substituted by milk and lemonade. He commends following the example of the London asylums and that of Krapeln, in Heidelberg, by prohibiting the use of alcoholics.

MACALESTER

**Multiple Paralysis of the Cranial Nerves Caused by Syphilis.**—Jacobson (*Centralblatt für innere Medicin*, March 2, 1895.) A man, aged twenty-nine, began to cough three months after the appearance of a hard chancre. Three months later, signs of cavities were discovered in the lungs, and the patient began to expectorate freely a greenish sputa, which, however, contained no bacilli. After about four weeks trigeminal neuralgia and double facial paralysis appeared, which



yielded to anti-syphilitic treatment. The condition of the lungs grew worse, and the author remained undecided as to whether the pulmonary lesions were of a syphilitic nature.

FREEMAN.

**Traumatic Atrophy of the Serratus Magnus.**—(Debedat (*Archiv. d'Electricité Medicale*, November, 1894).)

J. S., *et.*, 29, teacher, good family history. Physical examination shows nothing abnormal. He is well built and strong.

Two months before the beginning of the present affection he began the daily use of dumb bells, weighing about twenty-two pounds, which resulted in a fine development of the deltoid muscles.

One day, while exercising, he felt a severe pain in the neighborhood of the right shoulder blade, and at the same moment he felt the lower angle of the bone strike against the spinal column. The arm immediately fell until it formed an angle of  $45^{\circ}$  with the body. During the night the patient suffered a great deal of pain, and the next morning he was struck by the peculiar deformity in the scapular region. When the arm was brought forward the scapula projected very prominently behind.

A month after this the patient was first seen. Inspection of the back showed that the lower angle of the right scapula was more prominent than the left. The right arm could not be lifted higher than the horizontal position, but all other movements could be made with facility.

If he made the movement of pushing forward with his shoulder, the scapula immediately stood out from the surface of the body like a wing, and the lower angle strongly elevated. On muscular effort the scapula returned to its normal position. There was no pain in any of these movements. The electrical reaction of all the muscles of the scapular region was normal. The serratus magnus could also be seen to contract under the electrical stimulus. Extending downward from the lower angle of the scapula there is to be seen a slight furrow, or depression, barely large enough to hold the edge of the hand. This depression is undoubtedly due to the atrophy of the serratus magnus and taken in connection with all the other symptoms, makes the diagnosis of traumatic atrophy of this muscle certain. The traumatic origin of this affection is shown by the fact that the one muscle only is affected, though the patient is not in the least degree rheumatic or hysterical.

The treatment of this case consisted in daily local faradic applications, lasting for five minutes. At the end of three months he was completely cured. The appearance of the parts was entirely normal and all the ordinary motions could be made as well with one arm as with the other.

ROBINSON.

**Large Cerebral Tumor Without Headache and With Negative Ophthalmoscopic Examination.**—M. Pel. (*Gazette des Hôpitaux*, January, 1895.) A woman, aged 47, began to exhibit paralytic phenomena very slowly, the loss of power appearing first in the fingers of right hand, and gradually extending to the forearm. A year later the lower extremity became paralyzed, the intelligence and memory grew feeble, and an epileptiform attack occurred leaving a persistent trouble of the speech. Other attacks followed, and the mental condition grew worse. The diagnosis was doubtful, for there had been no headache, no lesion of the fundus of the eye, no nausea, vomiting or vertigo. By exclusion the case was admitted to be one of cerebral tumor. The patient was trephined over the motor centre for the upper extremity. Between the dura and the brain a tumor was found measuring seventeen centimetres in circumference. It was discovered to be a fibrous growth of the pia mater. Enucleation was easily performed, but the patient succumbed in a short time to heart failure. The parietal lobule retained its integrity, and, according to the author, this explained the preservation of the muscular and general sensibility.

FREEMAN.

**The Relation of Infectious Processes to Mental Disease.**—C. K. Mills (*American Journal Med. Sciences*, November, 1894.) The author refers to the report of Drs. Regis and Chevalier-Lavaure and to Dr. Hurd's paper on "post-febrile insanity," in which mental disorders (post-febrile) are regarded as developing from specific poisoning, from shock, from anemia and from nervous exhaustion. As subdivisions of specific poisoning are mentioned "the delirium of fevers, both intermittent and exanthematous, of pneumonia, of uræmic poisoning, the transient insanity of influenza, the mental confusion of multiple neuritis, the delirium of iodoform, salicylic acid and chronic alcoholic poisoning and the delirium of puerperal fever."

Regis and Chevalier-Lavaure studied selected cases of insanity to show whether there was excessive production or storing of products which should be eliminated. They studied blood, organic liquids and especially the urine, to find how toxic their qualities were during the various forms of insanity, and as compared with the same in health. The theory of the toxicity of intestinal contents was also referred to, also the possibility that general paralysis of the insane is due to infection.

The author then gathers the scattered opinions of others, trending toward the assumption of the "confusional" type of insanity as post-febrile; as toxic, as due to anemia and exhaustion following acute and infectious disorders, or (Korsakoff) as a toxemia due to a special poison developed in multiple neuritis, influenza and infectious disorders.

He refers further to a case of a febrile typhoid fever (Gerloczy) which was seemingly like acute mania. The description of "mental confusion" (Chaslin), as an intermediary between functional and organic insanity, is also brought into line. He refers briefly to multiple neuritis, myelitis and disseminated sclerosis, speaking both of the evidences of their infectious origin and of their interlinking with mental manifestations.

He refers further to a case of acute delirium with autopsy, showing incipient meningitis; to another in which no meningitis was found; to the studies of Mayberry into the effect of an epidemic of erysipelas upon mental troubles. Rasori's case of acute delirium with autopsy and careful bacteriological examination is fully detailed. He cultivated a bacillus, which injected into the rabbit produced death with symptoms of septicæmia in one and a half to six days. He also gives three cases occurring in Philadelphia in one of which Dr. Ball found a small bacterium.

Puerperal cases are held as furnishing a foundation for belief in infectious origin; the analogy of eclampsia is also referred to. His conclusions merely claim septic infection to be one cause of mental diseases, even while admitting the negative evidence found in autopsies and the but meagre bacteriological evidence so far obtained. PHELPS.

**The Role Played by Fixed Ideas in the Pathogenesis of Hysterical Polyuria.** Souques (*Archiv. Neurolog*, Dec., 1894).

The conclusions reached by the author on this subject are: (1) Continuous hysterical polyuria is much more common than has been supposed. Most previous observations of alcoholic, emotional and traumatic polyuria belong to this category. But it does not by any means encompass all the cases of polyuria *sine materia*. At least, the polyuria of hereditary degeneration forms a category distinct, and incapable of being brought into the hysterical category. (2) Hysterical polyuria is the consequence of a fixed idea of a urinous order. This idea fixes its origin in an anterior, a previous existence in the subject of an urinary trouble, a poikiluria, or hypersecretion (the result of an acute excess of alcohol, nocturnal incontinence of urine). (3) The idea inhibits, probably, the vaso motor centres of the kidney, which entails a renal vaso dilatation, that is to say, a polyuria.

J. C.

## Society Reports.

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### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, held at the New York Academy of Medicine,  
on Tuesday Evening, March 5, 1895.*

Dr. EDWARD D. FISHER, President, in the chair.

#### CHARCOT MONUMENT FUND.

Dr. C. A. HERTER, the chairman of the Charcot Monument Fund Committee, reported that \$640 had been received up to the present time.

Dr. ALFRED WIENER exhibited the brain removed from a boy aged fourteen years, who, eight weeks ago, had an attack of acute articular rheumatism. From this he recovered entirely, and was able to return to school. Eight days ago he began to complain of pain in the right occipital and frontal region and some pain in the right ear. The pain over the frontal region gradually grew more severe and afterwards became more pronounced on the left side. There were no eye symptoms at any time, and no discharge from the ears. A diagnosis of meningitis was made. The patient was seen in consultation by Drs. Sachs and Gruening, who confirmed the diagnosis, but the latter suspected a thrombus in the lateral sinus. The temperature became more and more elevated, and pyæmic in character towards the last, and the boy died in a stuporous condition on the seventh day. At the autopsy, made twenty-four hours after death a necrosis of the posterior surface of the petrous portion of the right temporal bone was found, with an acute meningitis on the opposite side of the brain. There was also a small thrombus of the lateral sinus, and the tentorium cerebelli was infiltrated with pus. The boy's mother stated that four months ago he had a suppurating otitis,

which seemed to have healed entirely, for up to four weeks before he was taken sick the ear had ceased to discharge and the perforation had closed. The advisability of an operation was carefully considered, but as there were not present enough symptoms to localize the lesion, and, therefore, warrant any such procedure, the idea of surgical interference was given up.

Dr. PHILIP MEIROWITZ exhibited a number of mounted cross-sections of the medulla, pons and corpora quadrigemina. The sections were taken from the normal subject and stained by Weigert's method, as modified by Pal and Vassale.

Dr. LOUIS F. BISHOP presented a case of œdema of the right upper extremity. The patient was a female, aged thirty-nine; married; no hereditary disease; eight brothers and sisters living; no previous serious illness; no cardiac, renal, or rheumatic history; has had seven children—five miscarriages; last child one year ago; no hysterical or other nervous symptoms; general health, excellent. Seven months ago, without any apparent cause, she noticed a swelling of the thumb of the right hand. Two months later she noticed swelling of the back of the hand. Quite lately the swelling has extended to the elbow. The swelling is worse at menstrual periods, and recently disappeared temporarily when in bed for a few days during an attack of influenza.

Physical examination: Heart sounds normal; lungs negative; right hand, œdematous; capillary circulation, more sluggish than on opposite side; pulse same as opposite side; no loss of power.

Examination of axilla, negative; no pain at any time. Dr. Bishop said he regarded the case as one of angioneurotic-œdema.

Dr. JOSEPH COLLINS said that Dr. Bishop's case, in most of its characteristics, did not agree with the symptoms that have been usually observed in these cases. Angioneurotic-œdema is evanescent, the swelling rarely lasting more than a few days, and does not pit on pressure. In this case there is slight pitting on pressure and an absence of the tense, hard skin usually seen.

Dr. C. L. DANA said that while this case did not correspond with the ordinary picture of this affection, yet there are cases of neurotic œdema which affect the limb in this way. All other possible causes of the œdema must be carefully excluded.

Dr. WILLIAM M. LESZYNSKY presented a woman who had an opening in the skull, in the left parietal region. The opening did not extend through the inner table. It was irregular in outline, with fissures running in different directions. There was no history of trauma, and the condition had existed from early childhood. Six years ago the patient began to suffer from epilepsy, the attacks being general in character. Dr. Leszynsky expressed the opinion that the loss of bone might be the result of a trauma in early life, followed by periostitis, or possibly the result of a syphilitic periostitis.

Dr. DANA said the deficiency in the cranium in this case was probably the result of a defect in osseous development, and he regarded it as one of the stigmata of degeneration which sometime accompany epilepsy.

Dr. L. STIEGLITZ exhibited a pathological specimen in connection with the report of the following case: The patient was a female, aged twenty months. She was first seen in November, 1894, when she presented the clinical picture of complete left hemiplegia, and paralysis of the right third nerve. The child was poorly developed. The left knee-jerk was exaggerated. Both pain and temperature sense were unaffected on the paralyzed side. The mother stated that six months before, she had noticed that the child did not use its left arm as freely as the right. The paralysis from that time on gradually increased, until the child gave up all attempts at walking. Three months before coming under observation the ptosis of the right eye came on, with divergent squint. A diagnosis of tumor, probably tubercular, in the right crus was made, although the possibility of glioma was considered. The following December paralysis of the left third nerve developed, indicating involvement of the left crus, and the child grew drowsy and apathetic. On February 17, 1895, there were twitchings, epileptic in character, on the paralyzed side of the body. Death occurred two days later. At the autopsy, which was made two hours after death, the brain was found to be large; the lateral ventricles dilated. In the right crus a tumor was found, with a cheesy centre. This spread upwards and forwards across the median lines into the left crus, involving the region of the nucleus of the third nerve.

Dr. B. SACHS referred to a similar case which he published in the *American Journal of Medical Science* some years ago.

The president, Dr. FISHER, said that the epileptic seizures in this case were of interest, as the tumor was so far from the cortex. They could only be explained on the ground that the impulses from the cortex were interrupted as they passed downwards to the cord.

A CASE OF TABES, ASSOCIATED WITH POST-  
HEMIPLEGIC ATHETOSIS AND UNILAT-  
ERAL REFLEX IRIDOPLEGIA (See page 294).

DISCUSSION.

Dr. SACHS said he was inclined to regard the case as one of cerebro-spinal syphilis of the tabic type. He would insist on that diagnosis even if no history of syphilitic infection was obtainable. One very important symptom which points in that direction is the dissimilarity of the ocular conditions on the two sides. The movements of the right hand, Dr. Sachs said, he hardly regarded as an athetosis; to call them ataxic or choreic he thought would be more correct. They were probably due to an irritating lesion of the capsule or the ganglia surrounding it. As regards the movements of the eyes, the speaker said that these lateral jerky movements which are frequently styled nystagmus are not to be compared with the rapid occillary movements we get in true nystagmus. He has noticed slight movements of this character in persons with tabes and in entirely healthy subjects.

Dr. HERTER said he still held to his original view that the case was probably one of multiple sclerosis. He did this chiefly because he believes that a certain proportion of cases of multiple sclerosis are of syphilitic origin. Notwithstanding the fact that it has been pretty clearly shown that this man has had syphilis, his symptoms correspond more closely to multiple sclerosis than to tabes. The man has probably had cerebro-spinal syphilis, and at the present time he has that sclerotic process going on which belongs to the later period of the disease. The changes that have occurred in his brain and cord are probably of a sclerotic, rather than of an inflammatory nature. Recent researches show that in multiple sclerosis the patches of degeneration scattered in an irregular way throughout the nervous system, are clearly related to the blood-vessels. Very often a blood-

vessel is seen in the centre of a patch and compound granular capsules and other evidences of subsiding inflammation are seen in the periphery of patches of undoubted multiple sclerosis.

Dr. HERTER said that in reaching a diagnosis in this case, he laid considerable stress on the nystagmus, which he believes is more commonly met with in multiple sclerosis than in tabes. This patient also has marked slowness and slurring of speech. There is an entire absence of ataxia in the legs, and he has never had any lightning like pains. The pupils are not the typical Argyll-Robertson pupils, and are more in line with those found in multiple cerebro-spinal syphilis than with those of tabes. It was stated at the last meeting that the man was impotent for a time: on questioning him about this he stated that there was a loss of sexual desire, but he was always able to have erections. The slight optic atrophy which exists is quite as characteristic of multiple sclerosis as of tabes.

Dr. J. ARTHUR BOOTH said that during his examination of the man, made a few days ago, forced flexion and extension of the ear produced a distinct occillatory tumor, which he regarded as a symptom of sclerosis. The man's penis bears a scar which he claims was produced by bringing it in contact with a hot stove.

Dr. FREDERICK PETERSON said that he was not prepared to uphold his diagnosis of multiple sclerosis in this case so positively, after hearing the thoughts and additions in the history as given in the latest accounts by Dr. Collins. The exhibition of the case had shown it at first as one of tabes with athetosis, supposing probably that the whole condition was one process. I am glad to note that he now believes with the rest of us that the condition is one of multiple lesions at any rate. Now the only symptom in the case which is particularly significant of tabes is the absent knee-jerks, which may be with him a physiological condition, or may be due to multiple sclerosis affecting the posterior columns.

The man has had no pain. He has no ataxia. He has no sphincter trouble. His pupils react to light, and the ciliospinal reflex is present on both sides, as I examined this latter reflex very carefully with the faradic current to determine that point. There never was any impotence in this case, according to the testimony of the patient, though Dr. Collins makes an important symptom out of a supposed impotence. On the other hand, we

have symptoms not at all related to tabes; such as a species of nystagmus, which, while not so typical a movement as we sometimes find in sclerosis, is still so marked that we may positively state that it is not the mere twitching of a neurasthenic condition, but a species of nystagmus due to organic disorder. There being no history of syphilis, it is difficult to concede the case to be one of general cerebro-spinal syphilis, a term which may be made to cover almost any obscure pathological condition in the central nervous system. The patient has multiple lesions, and the chief fact at issue can be positively stated to be that the case is absolutely not one of locomotor ataxia with athetosis, and that there is probably no tabes about the case at all, as he presents only one symptom of that condition.

Dr. G. M. HAMMOND said he also had examined the patient, and was ready to agree with all the speakers. He agreed with the statement that the man had post-hemiplegic athetosis; with Dr. Sachs' opinion that he had cerebro spinal syphilis, and with Dr. Herter that the lesions are multiple and probably sclerotic in character. The patient evidently had two distinct affections. He had had a cerebral hæmorrhage about fifteen years ago. The peculiar atheto-choreiform movements were the result of this lesion. Dr. Hammond did not agree with Dr. Collins in regard to the situation of the lesion. The fact that the patient had a post-hemiplegic mobile spasm indicated, in his opinion, that the initial lesion invaded either the striatum or the thalamus.

The second affection from which the patient suffered began about a year ago. It is an ataxia which differed but little in its details from the typical cases of that disease.

Dr. FISHER made a diagnosis of cerebro-spinal syphilis. He saw nothing which would indicate multiple sclerosis.

Dr. COLLINS, in closing the discussion, said he did not see how this could be regarded as a case of multiple sclerosis, unless our present conception of that disease be utterly changed. It is unnecessary to rehearse the different symptoms on which the diagnosis was based.

In reply to Dr. Peterson's remark that there never had been a period of impotency, Dr. Collins did not think it necessary to dwell upon this, for if the testimony of the patient and his wife is to be allowed, then impotency was present as stated. What Dr. Peterson meant by saying



that the chief fact at issue can be positively stated to be that the speaker was unable to grasp, as there is no fact at issue, and most of the gentlemen believed that the clinical picture was one of locomotor ataxia and athetosis. Unquestionably the man has multiple lesions, otherwise he could not have three distinct sets of symptoms; but multiple lesions do not by any means imply multiple sclerosis, for the latter is a clinical entity, having a well-defined symptom complex, and unless we are willing to depart from all previous ideas of multiple sclerosis this case can not be considered as in any way related to it.

### PALMUS.

Dr. LANDON CARTER GRAY read a paper with this title. He stated that within the last few years he has seen a number of patients with certain muscular movements to which the French authors have given the name "*tic convulsif*." They were first described in 1885 by Gilles de la Tourette, under the name, "A Nervous Affection Characterized by Motor Inco-ordination, Accompanied by Echolalia and Coprolalia." The details of this description are as follows: Motor inco-ordination is the first symptom, beginning gradually, and without apparently influencing the general health. The face or the upper extremities are first affected. It is first noticed that the fingers are alternately extended and flexed, or that the shoulders are raised. At about the same time the muscles of the face become implicated; there is often incessant winking, or the buccal commissures are actively drawn upwards and outwards, or there is contraction of the masseters, causing a grating of the teeth, or there may be projection of the tongue. The muscles of the neck may take part in the movements, the head being alternately flexed and extended. The lower extremities may share in the general disturbance, but the inco-ordination in them is somewhat different, in that the movements are not limited to isolated muscular groups, as is the case in the parts above described, but extend over the entirety of the muscles of one or the other member, and sometimes over both. The patient stamps his foot, stoops, or erects himself, or the movement may consist of a veritable jump. All these movements are very sudden and rapid, and may be accompanied by an inarticulate cry. At times this cry may be articulate, consisting of a word which varies in different

patients, and which was supposed to possess certain characteristics of an echo; hence the name echolalia. The coprolalia was regarded by La Tourette as pathognomonic, and consists of profane oaths or obscene epithets. The French authors had their attention directed to this subject by the descriptions of two American writers—those of Beard upon the Jumpers of Maine, published in 1880, and that of Hammond upon Miryachit, a similar disease of the Orient.

Dr. Gray said that the main symptoms of this affection are much more frequent than has been hitherto supposed, and he has ventured to coin for it a new name, that of *Palmus*, from the Greek word, *palmus*, meaning palpitation or twitching of the heart. The affection is divisible into the following types:

1. *Facial Palmus*. This constitutes the most frequent type. It consists of sudden, shock-like movements of lightning-like rapidity, causing a sudden wink or twitch of one cheek, occasionally of both, or of the brow. This series of movements is generally followed by a second series, weaker than the first, and sometimes by a third. In the majority of cases the winking is bilateral.

2. *General Palmus*. This consists of intermittent, shock-like movements of different muscles of the body.

3. *Acute Palmus*. In one case of this, coming under his observation the patient was a boy aged six years. He had then had the trouble about six months. His palmodic movements were very curious. For instance, when standing near a table looking at something, his chin would suddenly come down with a thump that would leave a black and blue mark; or, while standing quietly, his legs would give a sudden twitch, and he would be thrown violently to the ground.

4. *General Palmus, with Pseudo-melancholia*. Of this type two cases have come under his observation.

The diagnosis of *palmus* can be readily made. The only disease with which it is likely to be confounded is chorea, from which it differs radically. In chorea the movements usually involve the fibrillæ of muscles, and not muscular groups, either acting singly or in co-ordination with other muscular groups.

The prognosis varies according to the type. The author stated he has never yet seen a case of the facial or general variety cured; however, in none of those cases was he able to carry out prolonged and rigid treatment. The case of acute *palmus* was readily cured

under large doses of arsenic and rest in bed. One case of palmus with pseudo-melancholia was cured; the other was lost sight of.

Thus far no light has been thrown upon the pathology of this disease. As regards treatment, arsenic has a beneficial effect, but this is not of as long duration as the same drug has in chorea. In the facial and general varieties the arsenic alone will not answer, and in one case of generalized palmus with pseudo-melancholia it had no effect whatever. Hyoscyamine and hyoscyne have a good effect in some cases. Iron has been utterly useless; also the bromides. Galvanic electricity is of distinct benefit. In facial palmus he has seen good results from the removal of errors of refraction or irritative lesions of the naso-pharynx. Absolute rest is also indicated, particularly at the outset, and the general health of the patient should receive attention.

Dr. DAVID WEBSTER said that palmus appears to be an affection which is known under various names. About twenty-five years ago he first became acquainted with it under the name of blepharospasm. Later on it was called blepharo-facial spasm; and, still later, hemi- or bi-facial spasm, the latter when both sides of the face were affected. Then, again, it was heard of under the name of *tic convulsif*, and finally palmus. There are several varieties of the disease. The easiest kind of all to cure is that which only simulates the real disease, and is produced by a local irritation, such as foreign bodies in the eye, etc. Another variety is most common in school children, who are perhaps poorly nourished and use their eyes too much; sometimes a number of cases occur in the same school, which is probably due to imitation. These cases are generally curable. One such case under his observation was cured by the local use of atropine and proper glasses. In another case the symptoms disappeared after a congenital phimosis had been removed. A very severe and intractable variety of the disease is sometimes met with in the aged. Internally, he usually employs arsenic in the treatment of these cases. Conium has also been recommended.

Dr. G. M. HAMMOND said that about eight years ago he read a paper on this subject in which he reported a number of cases. In his experience, arsenic, even in toxic doses, rarely proves of much benefit. On the other hand, he has used conium with much success. He usually

begins with five-drop doses, which are gradually increased to twenty-five or thirty drops.

Dr. T. R. POOLEY said that two varieties of blepharospasm are usually recognized; the symptomatic, which depends on some eye lesion, the presence of a foreign body, etc., and the essential blepharospasm, which is entirely independent of such cause. In the first variety, if we cure the eye trouble we cure the blepharospasm. In both varieties the symptoms may manifest themselves in either the clonic or tonic form; in the former, the movements of the lids are momentary, while in the latter form, the lids may remain closed for a considerable period of time. In nearly all of these cases, pressure points may be found, generally in relation with the trigeminal nerve or some of its branches. The supra- and infra-orbital branches are often favorite locations. Such pressure points may also be found in the nose, throat and mouth, especially when carious teeth exist. In exceptional cases they are found in other and more remote parts of the body. When such a pressure is pressed upon, the movements cease like magic. The way to cure the disease is to attack the point from which the reflex irritation starts. Stretching of the nerve is recommended in severe cases.

Dr. GRAY then closed the discussion.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, February 25, 1895.*

The President, Dr. JAMES HENDRIE LLOYD, in the chair.

Dr. WILLIAM OSLER read a paper on

### NEURITIS DURING AND AFTER TYPHOID FEVER.

He gave an account of the cases which had been under his observation since the opening of the Johns-Hopkins Hospital; five cases of local neuritis; four instances of general peripheral neuritis involving arms and legs. Of the cases of local neuritis four occurred among about 350 cases of typhoid under treatment, and one was a dispensary case. He described first the condition which has been referred to as "tender toes" in typhoid fever, which was possibly also a peripheral neuritis.

In two of the cases the local neuritis developed at the height of the fever; in three, after the temperature had become normal. He spoke of the difficulty of diagnosis between some of these cases of localized peripheral neuritis after typhoid and poliomyelitis.

Of the four cases of multiple neuritis none were under treatment in the hospital during the primary illness. All four were in males: one a boy of nine, one a boy of ten, and the other two in young adults; one case presented a very marked relapsing feature. In these four cases there was double wrist and foot drop, with atrophy; in all of the cases there was marked pain in muscles. There was no trophic disturbances, no involvement of the bladder or rectum. Two of the cases which were under observation in 1893 have recovered completely. The two cases which were under observation in the autumn of 1894 have improved very much, particularly in the arms, but still have foot drop and the steppage gait.

## DISCUSSION.

Dr. WHARTON SINKLER.—As Dr. Osler remarks, it is extraordinary that so few cases of neuritis following typhoid fever have been reported. At the same time, we must remember it has only been within the past ten or fifteen years that multiple neuritis from alcohol and other causes has been reported. The various forms of paralysis after typhoid fever that have been reported have no doubt been most commonly due to poly-neuritis.

I have seen one notable case of neuritis following typhoid fever in a girl of thirteen years. She had a severe and prolonged attack of typhoid fever with a relapse, and during the relapse, the symptoms of neuritis manifested themselves. There was tenderness of the extremities, followed by complete loss of power. When I saw her several weeks later, there was great wasting of the lower extremities, very little hyperæsthesia or tenderness of the nerve trunks, but there was loss of power, almost complete, and strong contractures of the legs at the knees. Persistent massage and stretching failed to relieve these contractures, and when the muscular power had been restored the ham-string tendons were cut, the limbs straightened and the patient completely recovered the power of walking.

This case is of interest as Dr. Osler stated that he had not seen paraplegia from neuritis.

Dr. FRANCIS X. DERCUM.—Dr. Osler's paper presents a number of interesting points. For instance, in one of the cases related there was, after partial recovery of power, again a subsequent loss. We see this now and then in other forms of neuritis, especially if the patient exercises at too early a period. It was recently observed in a case of beri-beri at the Philadelphia Hospital in which there was marked recovery with subsequent relapse after the patient had been about.

With regard to the points of differential diagnosis I think that to the tenderness and pain we should add hyperæsthesia. This, if present, would throw the weight of probability in favor of multiple neuritis rather than poliomyelitis.

I am a little doubtful with regard to the point that total extinction of faradic reaction in poliomyelitis is permanent. In a limited number of cases of infantile paralysis I have seen a return of electrical reactions where none were obtained at first.

I think that one reason why cases of multiple neuritis after typhoid fever are not more frequently reported is because the symptoms are frequently not as typically developed as in toxic neuritis from other causes. I have seen a number of cases of a mild type, but none that were as typical of multiple neuritis as those due, for instance, to alcohol.

It seems to me that in the differential diagnosis between poliomyelitis and multiple neuritis the age of the patient is an important element. Acute poliomyelitis is rare at those periods of life when we are apt to have typhoid fever. I think that in cases otherwise doubtful, we should lean strongly to the diagnosis of multiple neuritis, if the affection occur in adolescence or adult life.

Dr. JAMES TYSON.—In a fairly large experience with typhoid fever, I have not met with cases of secondary multiple neuritis. The question of the special etiology appears to me of interest. I think that Dr. Osler did not refer to this. As the affection comes on during convalescence, after the fury of the disease is spent, it seems difficult to say what the special cause is.

We are all familiar with the muscular pain in the early history of typhoid fever. I should like to know if there can be any connection between these two conditions, the extreme pain in the early stage and the neuritis at the close.

Dr. C. S. POTTS.—I can support what Dr. Dercum has said in regard to faradic contractility in some cases of poliomyelitis. I recall one case treated at the University Hospital where there was total extinction of faradic contractility, with subsequent return.

Two years ago I reported a case of multiple neuritis following typhoid fever. The case was referred to us by Dr. Musser. In the third week of the disease violent pain was experienced in both legs, and when the patient was allowed to get up there was found to be more weakness than would be expected after the attack of typhoid fever. When the patient was referred to us there was hyperæsthesia over the calf muscles of both legs, tenderness in the popliteal spaces and reactions of degeneration in the tibialis anticus and peroneal groups. Under the use of massage, electricity and strychnia continued for several months, the patient recovered. This case I think would be regarded as belonging to the paralytic type mentioned by Dr. Osler.

Dr. G. BETTON MASSEY.—The value of the faradic current in showing the difference between multiple neuritis and poliomyelitis would lie partly in the period of return of contractility. It would be late in poliomyelitis when it did appear, and would appear early in multiple neuritis.

Dr. M. V. BALL.—The pain in the feet after the use of the Brandt method, to which Dr. Osler has alluded, was observed at the German Hospital in cases treated after this method. The nurses frequently found it necessary to roll back the bed clothing so as to leave the feet exposed.

Dr. JAMES HENDRIE LLOYD.—I have been much interested in the study of a case of peroneal muscular atrophy following typhoid fever, a report of which was made to the College of Physicians in January. In that case I was struck by the resemblance to the peroneal type of muscular atrophy reported by Charcot and others. Sir James Paget called attention to the fact that after typhoid fever there is apt to be an involvement of the peroneal nerve. It seems to be a coincidence that these muscular atrophies of a peroneal type have been noted as following infectious diseases. In my case the atrophy was most marked in the peroneal group of muscles. It was followed by contractures which are persistent. In this case there was not much muscular pain; there was no anæsthesia and no typical reactions of degeneration.

I think that we sometimes take a too restricted view of the pathology of these cases. Some cases are due possibly to poliomyelitis, but some may be due to affection of the muscular tissue itself. According to the researches of Zenker and others there are grave changes in the muscles during typhoid fever. This is a point which has been too much neglected.

Dr. OSLER.—The relapsing feature in one of these cases was of great interest and seemed to clinch the diagnosis of a peripheral against a central lesion. It is well known that certain types of neuritis are characterized by this tendency. Many of the cases of paralysis after typhoid are due to poliomyelitis. Indeed, Gowers makes the statement that after no acute fever do we see this spinal affection so frequently as after this disease. I have never met with an instance, whereas I have seen several cases of poliomyelitis with permanent and per-



sistent atrophy of the muscles after measles and scarlet fever.

I did not touch upon the etiology of post-typhoid neuritis because we do not know anything about it; at any rate, not enough to speak with certainty. As in diphtheria, it would appear that it is more commonly a late lesion. The observations of Pitres and Villard would indicate that it is not a very uncommon event, as they have found the anatomical lesions in cases which presented no signs of neuritis during life.

Dr. LLOYD has raised an interesting point in some of these cases as to the presence of a myositis rather than a neuritis. In two of the four cases in which, during the height of the fever, acute local symptoms supervened, which I regarded as a neuritis, there were swelling and tenderness of the muscles. We must remember, however, that tenderness of the muscles is a very special feature of many cases of multiple neuritis (particularly as pointed out by Wilks) of the alcoholic form.

An important matter in this group of cases relates to the prognosis, the hopefulness of which has seemed to me very remarkable. Two of the cases of multiple neuritis have already recovered, and the other two have shown improvement enough to warrant the expectation of a similar good result. This satisfactory feature of multiple neuritis is not, however, confined to the post-febrile forms.

Dr. M. V. BALL read a paper on

### SOME CASES OF SEXUAL PERVERTS.

And described the mental characteristics of eight male sodomites, who were serving terms in the Eastern Penitentiary. They had either been sentenced for this crime or had confessed to habitually practicing it.

In all of them feminine traits were strongly developed, though their physical appearance was masculine. Three of the eight were negroes.

In the majority of cases the habit was commenced very early, even before puberty, and in four cases was practiced in houses of prostitution.

The individuals solicited trade on the public streets as female prostitutes do.

Delusions or other signs of insanity are not present.

There are two classes of sodomites, those who make a profession of it and who use all the arts of a prostitute, and another class who possess the traits of the sexual pervert, but only commit the act when under peculiar circumstances, as when in prison, in the army or navy, or in close association with men.

In the cases studied, desire for the female sex was wanting. Pederasty was never practiced.

#### DISCUSSION.

Dr. FRANCIS X. DERCUM.—It seems to me that when we know more about this subject, we shall find that there are really two kinds of sexual perverts, those in whom the practice is acquired in early childhood and is kept up, and those in whom it is a symptom of general nervous degeneration. It is interesting to study these cases for signs of physical degeneration; for instance, about the teeth, the ears, the digits, etc. Measurements of different portions of the body should be made and feminine characteristics sought for. The subject seems to be an interesting one from that standpoint and would repay more detailed study.

Dr. M. V. BALL.—Investigations, such as Dr. Dercum has alluded to, have been carried out, and in some cases there have been found degenerative stigmata, but in many cases there have been no signs of physical degeneration. While most of these cases give a history dating back before the development of the sexual passion, yet we can not believe all their statements. Many of them are prone to boast of their achievements. Many people are sent to prison for sodomy who are not sodomists, but, who in a condition of drunkenness, have been caught in bestial acts. These cases show none of the regular characteristics of sodomists and are of interest to the ponologists.

Adjourned.

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OF  
**Nervous and Mental Disease.**

**Original Articles.**

RELATIONS OF THE CEREBRAL CORTEX TO  
SENSATION.<sup>1</sup>

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EVERY marked advance in our knowledge of the structure of the nervous system is of interest to the practical physician because of its bearing upon the understanding of nervous function, as this in turn offers the only prospect for the correct interpretation of nervous derangements. In comparison with the histological investigation of nervous tissues the minute structure of any other tissues, even the most complex, is simplicity itself. We cannot, therefore, appreciate better what recent progress in this respect amounts to than by noting at what a stand-still investigators found themselves but a few years ago when the most patient dissection and microscopic observation seemed to terminate, as far as the gray matter was concerned, in a totally unravelable maze of minute, soft, interlacing threads, which, especially in the cerebral cortex, seemed to increase out of proportion to the cells, but in direct proportion to the presumable rise in grade of function. An insurmountable obstacle, therefore, purely physical in its nature, appeared to forbid any prospect of further penetration to the ultimate facts of nervous anatomy.

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<sup>1</sup> Read before the New York Neurological Society, April 2, 1895.

On the other hand, the knowledge so far gained seemed to point to the following conclusions:

1. The chief element in nervous matter is the ganglion.
2. The chief element in a ganglion is its cells.
3. The nerve fibres are simply channels of transmission to and from cells.
4. That certain associations of ganglia made to act together by commissural connecting fibres, constituted "nerve centres" endowed with special functions.
5. That these functions were carried out by corresponding tracts of nerve fibres.
6. That the nerve fibres themselves either made anastomoses between cells or were lost in an intercellular granular matrix.

It is not needful for me to describe to such an audience as this the methods by which the slenderest threads of nervous protoplasm have been made separately to show themselves, and thus to be tracked, however devious their intertwining and ramifications with other fibres. Golgi's staining, aiding also the further prosecution of Fleschig's developmental method, and Marchi's staining of degenerating tracts, have carried on to a point where some conclusions seem well nigh demonstrated, which, as we compare them with the previous views, cannot but modify in more than one respect our whole conception of nerve function. These conclusions in brief are:

1. That the anatomical unit of the nervous system is neither a ganglion nor a cell.
2. That a nerve fibre is not simply a channel of transmission.
3. That the anatomical unit of the nervous system is a cell with its process or processes, these together constituting an anatomical entity which has no direct or anastomatic connection with other nervous cells or processes. All relations of this anatomical nervous unit with others are physiological relations through proximity or contact, and not by fusion of fibres.
4. Therefore, the origin and seat of nervous function are not restricted to the cell, but rather are so far shared by its processes that it has even been surmised that the cell has for its chief office to maintain the nutrition of the processes, and to influence the direction, rather than solely to originate function. Hence, the development and elaboration of function would seem to be dependent

more upon the development of the protoplasm of the processes than upon the development of the cell itself.

These conclusions are based upon the consideration of the anatomical unit itself, viz., a nerve cell and its processes, to which Waldeyer has given the name neuron. Considering how desirable it is to have our conceptions kept definite as to the fundamental facts of nervous structure and function, it is a pity that Waldeyer's term has already been modified in its applications by various writers. Thus Schäfer restricts "neuron" to the axis cylinders processes of cells, while to the branching processes he applies the term "dendrons." As the most important proposition, regarded from the physiological point of view, is that the cell and its processes always constitute a concrete unity, or a nervous system, so to speak, by itself, it would seem best to keep some term simply to designate this fact, as Waldeyer originally proposed with "neuron," and which is good enough, pace some etymologists in Greek, for this purpose; while other terms might be devised for the neuron's constituent parts.

One of the chief deductions from these investigations seems to me to be a more definite conception of the plasticity of nervous matter. Instead of being confined within the exclusive limits of a nerve cell, we appear to have an active expanse of nerve matter and of nervous function reaching to the extremest peripheral distribution on the one hand, and similarly profoundly operative around the inner cells on the other, constant reaction and interaction between the terminal arborescent expansions at both ends taking the place of simple centric transmission in either direction.

Some interesting questions, therefore, now arise.

1. If no cell in the nervous system exists without process or processes, and as each cell with its process or processes constitutes an anatomical unit, by what means do these units become so interacting as finally to become organized to perform certain special functions together?
2. To what extent does such organization of special collections of neurons merit the term "nerve centre" being applied to them?
3. Does precedence in such organization in order or in time of development, as in the spinal centres, give greater fixity to such organization so as to afford justification for applying the term centre only to such seats of function, while to others of later development, as in the cerebral cortex, this term ceases to have

the same application on account of the greater plasticity of their elements, allowing more latitude in function according to the temporary training of individual life rather than the permanent hereditary organization of the spinal centres? The specially trained regions of the cortical centres in man, both afferent and efferent, connected with the function of speech and which are neither original nor hereditary in their capacity for speech, being illustrations in point.

To the first question, how does any given collection of neurons become so organized to work together as to perform a specific function, the recent advances seem to emphasize the fact all the more, that recurrent afferent impression on the extremely plastic peripheral termini until they become habitual, or normal in the etymological sense of the term: that is, according to rule, are the primary and essential sources of nervous organization from the simplest to the most complex examples of such organization. As the simple sensitive ectodermal cell in the hydra with but a single process proceeding direct to a contractile fibre within, is one of the earliest manifestations of nervous function, so each step in more complex organizations seems but a further and further development of the effects of afferent impressions. Afferent habit, in other words, is the origin of every nervous habit, *i. e.*, special function, whether in single neurons or among associated neurons. In time, habitual impressions, followed by corresponding habitual reactions, become opposed to non-habitual impressions, and thus give origin to the great function of inhibition, without whose constant influence no permanent organization would be possible. Under the old view it was the inner cells which became thus organized in response to afferent impressions which were radiated from cell to cell through commissural fibres, which thus became established tracts of intercellular impulses. The new view gives us more reason to apportion a larger share to the plasticity of peripheral nervous protoplasm. In fact, it is not easy to limit the degree of that plasticity for acquiring function. We seem to have, for example, in the nervous mechanism of the earthworm a simple division between the sensitive cells of its epidermis and the motor cells of its axis, with but a single connection between. And yet so varied has the peripheral protoplasm become in its susceptibility, that the worm, with neither eyes nor ears, yet evidently reacts both to light

and to sounds. From considerations to be adduced soon, it does not seem illogical to infer that cortical gray matter is likewise a plastic "peripheral expansion" and that the only relatively stable and non-plastic centres are the spinal centres.

But does any different principle or law of development enter into the organization of the spinal segments of vertebrates? We do not see the slightest reason for such a supposition. Nor would there be any occasion for such a supposition but for the gradual development, as we ascend to the upper segments of the cord, of newer and special manifestations of the afferent apparatus which oblige us to apply to them the elastic and portentous term sensation, not as a modification of afferent impression simply, but as introducing us to the boundless realm of consciousness and psychics. But where and when does an afferent impression become truly "sensory" and thus pass into a sensation and that to a definite consciousness? Does the entrance of this psychic element imply any new arrangement of nervous elements in any particular region or place, which, therefore, may be called the seat of sensation, or the area of sensation?

The answer to this question should not be sought in an examination of the functions, local or general, of the cerebral cortex, for that would be attacking the problem by dealing first with much its most complex aspect. Instead it should be insisted upon that experimental research does not oblige us to go any higher than the spinal system proper to locate the whereabouts of sensation, whether of a general or of a special character. We approach the confines of sensation instead by gradual stages through a series of wonderfully organized collections of spinal neurons, which have learned to work together so thoroughly that they have all the perfection of complete consciousness and purpose in that working. The headless body of a frog can be made to behave as if the head was still there. It is to be particularly emphasized that no cerebrum is needed to fill out all the requirements that can be asked in the way of evidence that sensation, and action dependent upon sensation, are already present. This is proven by removing the cerebrum and noting what functions remain.

To begin with, osseous fishes, such as the carp; when the ganglia which in them correspond to the cerebral hemispheres, are removed, there is little, if anything, to

distinguish them from normal animals. They maintain their normal attitude and use their tails and fins in swimming with the same vigor and precision as before. Lest this be considered as only equivalent to the unconscious spinal adjustments in the body of a decapitated frog, the experiments of Vulpian and Steiner with the fishes show not only that they are still able to see, but that they also can find their food. If worms are thrown into the water in which they are swimming, they immediately pounce upon them. If a piece of string similar in size to a worm be thrown in, they are able to detect the difference and they drop it after having seized it. They even to some extent distinguished colors: for when one red and some white wafers are thrown into the water, the fish almost invariably selects the single red one in preference to the white. So also with the frog. In Schrader's experiments, frogs which recovered from the ablation of their cerebral ganglia behave then like full brained frogs under like circumstances. They crawled under stones or buried themselves in the earth at the beginning of winter, and after the period of hibernation was over they came out and diligently caught the flies which were buzzing about in the vessels in which they were kept. Rising in the scale, Schrader's and Von Recklinghausen's experiments are too well known to detail about birds, who, without cerebral ganglia not only avoided obstacles in their path or in their flight and would fly from one place and alight securely on another, but were quick to ruffle feathers and to show fight when put in the proper position with other birds.

I do not see, indeed, how the position of some neurologists about the exclusive relations of the cerebral cortex to sensation, which we shall allude to presently, can be fully maintained in view of the published experiments of Goltz upon dogs, except by showing that Goltz's dogs differ from all other dogs. No number of negative results will disprove the positive results which Goltz reports and which he maintains are the only trustworthy observations yet published in their bearing on the questions raised, because the dogs survived the operation long enough to have all the vitiating effects of the primary injury pass off. In his seventh communication *Archiv für Physiologie*, Vol. v., p. 570, Bonn, he details his success in removing the entire cerebrum without killing the dog. The animal was kept alive for eighteen and one-half months, and post-mortem examination showed



that all the cortex had been removed, except from a small portion of the basal surface of the top of the temporal lobe. In addition to the cortical lesion, a large part of the corpora striata and thalami and a small part of the mid-brain had been destroyed. The small remnant of the temporal cortex not actually destroyed was, of course, practically removed, since all its peripheral connections had been interrupted. This animal slept naturally and could be awakened by loud noises or by handling. Strong, painful stimulation of the skin caused him to bark or growl, or even to snap. In making this last movement he turned in the proper direction to left or to right. He was able to maintain his equilibrium properly when one foot was placed on a falling trap door. He was able also to move round on three legs. He also closed his eyes when a bright light was thrown upon them. He ultimately learned to eat and drink provided his nose was brought into contact with the food. That he would also taste was shown by his invariably rejecting food moistened with quinine, after chewing it for a while. He also showed unmistakable signs of hunger or of thirst when deprived for some time of food and drink, and the eagerness with which he took the food when it was afterwards given to him was full proof of his hunger sensations. Goltz, therefore, comparing his dogs with the pigeons of Schrader and the fishes of Steiner, maintains that the mechanism of the cord and portions of the brain posterior to the cerebrum are approximately identical in all higher vertebrates, and that his dog without a cerebrum compared with a normal animal showed defects only in the manifestation of intelligence, memory, reflection and understanding: that is, in what are called the higher psychical functions.

In his other experiments the same conclusions are just as fully borne out, though in none did the animals survive as long as this one. In view of these facts, it certainly is difficult to deny that in these lower portions of the nervous axis, speaking relatively to the cerebrum, we have all the conditions which can be supposed necessary to constitute true nervous centres or seats of sensation. We have undoubted manifestations of the presence of every variety of sensation, tactile, muscular sense, sense of pain, vision, hearing, taste, and finally the visceral sensations of hunger and of thirst. Moreover, from the very limited areas of gray matter in these parts compared with the great expanse of the cere-

bral cortex, the conception of definite and strictly limited collections of neurons subserving special functions for which they have been permanently and hereditarily organized as the "centres" of such functions, is much clearer than can be predicated of any portion of the outer cerebral layers.

On the other hand, it cannot be denied that both experimental and pathological observations show that certain areas of the cerebral cortex have special relations to both sensation and motion. The question now is what is that relation? Is it a transfer of sensation from a lower to a higher seat, or is the seat of sensation proper still in the lower level, while the corresponding cortical function is for the cortical interpretation, so to speak, of the activity of the lower centres? It is evident that if a sensation has anything to do with a cerebral process that then it will have other elements in it than if it partook only of elements in the processes whose seat was in the ganglia of Goltz's dog. A cerebral process of sensation, in other words, would certainly include a cerebral recognition of the nature and character of the sensation as well as the sensation itself. By the influence of long habit, however, it would become very difficult so to analyse this cerebral act of sensation as to separate its cortical from its non-cortical elements. A sudden suspension, therefore, of cerebral sensation would involve so much loss of its habitual accompaniments as to appear to amount to complete suspension of sensation itself. A complete abolition of sensation, however, would be a permanent effect, if sensation had its true seat in a cortical area. That it is not so is Goltz's contention. All he asks is that time enough be allowed after any lesion in a cortical area to show that such lesions, however extensive or complete, nevertheless do not abolish either sensation or motion.

The readiest way it seems to me whereby to arrive at practical conclusions about the relations of cortical areas to these functions is to take into consideration, first, some general facts about the anatomical organization and sequence of development in function of the cerebral cortex itself. In foetal life the cells of the layers of the cerebral cortex are both small and closely packed together. The cell bodies up to a certain age (Andriezen)<sup>2</sup> seem to grow in size, but that limit is soon reached

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<sup>2</sup> *Brain*, 1894, p. 639.

while the animal is still young. But meanwhile the cell bodies certainly seem to get further apart during early life, much more so than their slight increase of size would be adequate to account for, and which, therefore, used to be explained by various authors as due to increase of blood-vessels, of neuroglia or of ground substance. But recent discoveries demonstrate that this phenomenon is correlated not with the growth of non-nervous structures pushing the nerve cells further apart, which cells, by the way, seem to cease to grow in number after early life, but by a progressive increase in the intercellular protoplasmic nervous dendrons or fibres of the cells. This growth in the extent and complexity of connections of the protoplasmic processes of the brain cell, both basilar and apical, causes the cells which in intra-uterine life lie so closely packed as to touch each other, to separate more and more widely until in the adult they seem to have become relatively quite sparse. We find in this anatomical fact certainly a strong presumption that the cerebral cortex grows in functional capacity by greater increase and elaboration of its nerve matter, though not of its cellular elements. Corresponding to this capacity for growth of its intercellular elements appear certain developments of functions in the cerebral cortex which are markedly different in genesis from anything which can be predicated of the special centres, in that they are newly acquired during individual life and are thus incapable of hereditary transmission. This fact so significant of the true relations of the cerebral cortex to function, is well shown in the acquisition by certain cortical areas in man of such highly specialized functions as sensory and motor speech. What the plasticity for acquiring function must be in this case is further shown by its localization in only one hemisphere, evidently through habit alone, determined in the first instance by the greater use of the right hand in gesture language. The corresponding areas in the other hemisphere, not being used, remain functionless in this respect. The phenomena of word-blindness, for example, following upon a lesion of a particular anatomical area is certainly a sufficient demonstration of the educability rather than of the inherent capacity of cortical matter for subserving function, because in this case this function was acquired long after all other cerebral functions had become settled and organized. A person learns to read and write, or rather his particular cortical

area, therefore, learns to do so, not by reason of any particular endowment of that area for that purpose, but solely because it has been made to learn it by repeated afferent and specifically guided impressions coming through the eye. No doubt also that later still in life, the protoplasmic dendrons of the central arborescent terminations, in the cerebrum, of the tactile nerve, become just as specially organized in the blind who learn to read by the raised letters provided for them.

The bearing of these considerations on the origin of the relations of the Rolandic areas to sensation and to motion is obvious. They show that we need not predicate any original or necessary connection of these areas with either sensation or motion. When a child learns to use its hands or its feet, it is just as likely while doing so to be developing special cortical areas for sensation and for motion, which may not originally have been endowed with those functions, as it afterwards educates certain areas of its left hemisphere to hear words, others to see words, and others still to express words. Certainly its attempts to learn to use its hands and its feet require just as protracted, repeated and laborious effort as its subsequent efforts to learn speech, which latter end in particular modifications of its cerebral cortex. Why, therefore, may not the sensory and motor functions of the Rolandic convolutions be as much acquired as the sensory and motor speech convolutions acquire their functions?

If this be so, we then can perceive that the educability of the cortical gray matter is such that probably in no two persons are the special areas corresponding to special functions exactly the same in extent. In one the respective areas may considerably overlap the boundaries of the equivalent areas in another. Such overlapping, indeed, probably has been already demonstrated in the motor areas, for Sherrington has found degeneration fibres in the pyramidal tract extending as far as the lumbar region following upon a lesion of the so-called cortical arm centre. On the other hand, the intimate blending of afferent or sensory, and efferent or motor elements in the Rolandic area is just what we should expect if these parts have been educated to their business. Motion is here immediately dependent upon, and directed by sensation as nowhere else in the nervous system. Respiratory (spinal) movements may be rhythmical, but phonation (cerebral) movements must be

immediate, and so with the rest. On the other hand, in the cortex itself the areas related to the sensory functions of sight and of hearing might relatively be considerably removed, as we find them, from the motor structures. So also the close blending of the functions in the Rolandic area is illustrated by the common occurrence of the sensory phenomenon of allochiria along with motor paresis.

We conclude, therefore, that, properly speaking, there are no centres for either sensation or motion in the cerebral cortex. Such terms should be restricted to the structures which correspond to Hughling Jackson's lower level of the cerebro spinal system. The cerebrum acquires its functions, however related they be to sensation or to motion, by education rather than by intrinsic endowment, and this fact accounts both for the comparatively indeterminate boundaries of its functional areas, and for the temporary nature of the effects of lesions in them. As Waller remarks, "The clinical history of a dog or of a monkey having suffered a removal of some part of the Rolandic area altogether negatives a strict limitation of function, and at most suggests its local concentration. Even clumsiness is not apt to be permanent. We thus picture the cortical organ in a semi-fluid state of differentiation, still variable by new instruction, rather than a petrified and invariable collection of specialized organs tied down to particular functions."

*The death* of B. Hack Tuke is announced in recent journals. There is probably no one name so well known to alienists, and again there is no one name among the lists so prominent for kindly, benevolent, uncritical spirit of helpfulness. His life was one impaired by ill health, and his writings are therefore more remarkable from this fact. The Dictionary of Psychological Medicine, which bears his name, will be a fitting monument to carry his fame and influence to his successors. The patient and continued unselfish labor which is represented tells clearly enough the spirit of the man.

## THE INFLUENCE OF HEREDITY ON IDIOCY.

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THROUGHOUT the history of education and of medicine no point has so engaged the attention of the philosopher and the scientist as the influences of environment and heredity. These influences upon our physical being, repeated through successive generations, resolve themselves into law, a law as unalterable as that of the Medes and Persians—a law founded upon fundamental truths and verified in the experiments of physiological science.

The aim of education and medical science is one, viz., to secure to the individual a sound mind in a sound body, so that all that touches one must find its prototype in the other. In this more than ought else, if one member suffer, all the members suffer with it.

This law of balance and proportion we recognize in the forces of nature, in the applied arts and sciences, and we are quick to note the evil effect of the least deviation from this rule.

In a nature so complex as man—a mechanism so finely ordered—none deny that the same principle is but intensified.

Here, therefore, in natural sequence, the *psychological* follows the *physiological* research, may rather join hands in this effort to restore health to the mind or to prevent degeneration; and, acknowledging the sins of the father transmitted in the *body* to the third and fourth generation, would fain seek in the springs of life itself the influence of heredity upon the mind—the secret and cause of idiocy. Most arrogantly we call ourselves “the heirs of all the ages” in all good gifts, but we are slow to acknowledge the serpent’s trail in our Eden. Our vanity is sensitive, and recoils naturally from the thought of blemish in our own—physical or mental—

ancestry, either ignoble or unfortunate. We do not, and we will not, except under protest, receive the law of heredity of ill.

Convinced that it is logical, still we seek proof in statistics. The gathering of statistics under these circumstances is difficult. Parents and friends are loath to accept such evidence, and, actuated by false shame, still more loath to impart information, especially when there is neurotic taint. The physician in many cases, feeling that he is betraying a confidence, keeps no record, and the literature on the subject becomes, therefore, most meagre. Thus, when we begin to sift the matter as we should like, we are frequently unable to verify our conclusions. So in many cases the causes of idiocy remain unexplained, closeted within the secret recesses of nature.

But the field of research, with all its difficulties, is broad and is practically unexplored. Before going into statistics, however, let us consider for a brief space opinions of deep thinkers on this subject.

Mercier says: "The first and most fundamental law of heredity is that *every attribute of the parents tends to be inherited by the offspring*. Inheritance is the law, non-inheritance the exception." You will observe that he does not say they *are*, but that they *tend* to be inherited, and the question therefore resolves itself into this, either the offspring does or does not resemble its ancestors. If the former, then there is an hereditary law; if the latter, there is not.

Strong as this is, we find in Montaigne a stronger thought: "Is it not marvelous that this drop of seed from which we are produced should bear the impression not only of the bodily form, but even of the thoughts and inclinations of our fathers? Where does this drop of water keep this infinite number of forms? and how does it bear these likenesses through a progress so hap-hazard and so irregular that the great-grandson shall resemble the great-grandfather?"

Blandford says: "Two laws of nature are concerned in the production of these phenomena. One is that peculiarities and abnormalities are apt to recur in descendants for many generations; the other, that there is always a tendency to return to the type of health in beings which have sufficient vitality to perpetuate their existence and carry on their race for successive generations."

We do not always produce an idiot from an neurotic family.

Again, one or even more of a family may be insane or idiotic, the others normal, showing the taint concentrated, due possibly to surrounding conditions and temperament of parents at time of conception combined with prenatal influences upon the mother.

Thus every idiotic child bears the mark of some inherited tendency from some ancestor.

I will here cite a case among my own patients, where a male idiot born of imbecile parents possesses the face and form of the mother with the disposition of the father, a type of both physical and moral imbecility.

Neuroses are frequently interchangeable in transmission from generation to generation, and there are two great laws—the reversion to the original healthy and perfect type when the taint is less noticeable in generations until it at last is not found at all; or it becomes more pronounced, and a simple nervous disease may appear in successive generations in the form of a pronounced neurosis.

From Ireland<sup>1</sup> we have the following: “Of all known diseases perhaps idiocy is most frequently propagated by heredity.”

Moreau, of Tours, affirmed that heredity was found in nine-tenths of his cases, but gave no statistics.

According to George Wallington Grabham,<sup>2</sup> heredity is the chief agent in the production of idiocy. Although in his statistics he collects but 18 per cent., he is convinced that the taint exists in a far greater proportion.

Dr. Shuttleworth, in a recent personal letter, writes: “My opinion is that heredity plays a very important part in the production of idiocy, though we cannot prove the frequency by statistics so completely as the fact is impressed on one’s mind by intercourse year after year with the relations of acknowledged idiots and imbeciles.

“Taking only the friends’ statements, we should be much misled, and for that reason I declined when at the Royal Albert Asylum, to publish each year any table of causes. . . . But as one got to see more of the relatives, one gradually accumulated a considerable body of evidence, and this has been summed up and compared with Dr. Beach’s experience at Darenth.” These observations are as follows: The most frequent

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<sup>1</sup> On the Diagnosis and Prognosis of Idiocy and Imbecility, p. 1.

<sup>2</sup> Remarks on the Origin, Varieties, and Termination of Idiocy.



hereditary factor is phthisis, which is found existing in 28.31 per cent.; inherited mental weakness, 21.38 per cent. (16.47 per cent. family history of insanity, 4.69 per cent. family history of imbecility), whilst in 20 per cent. there was a family history of neurosis.

Fletcher Beach subsequently made some further studies alone at Darenth, and found heredity in 76 per cent. of these cases.

In 1856 the Legislature of Connecticut<sup>3</sup> appointed a commission to investigate the cause of idiocy. The questions propounded were as to whether there had been idiocy, insanity, blindness, deafness, epilepsy, or any other defect, mental or physical, in the immediate or collateral family of the idiot.

The commission reported that out of 164 cases it found 70 where heredity was undoubtedly the cause; in 10 cases idiocy in the parents; in 6 in the various relatives; 6 insanity in parents; 8 insanity in relatives; in 8 epilepsy of parents or relatives; in 2 blindness; 1 melancholia in father; 13, dementia following insanity in relatives; and in 16 one or more of the defects mentioned, but not designated.

In the census of 1873, taken at Berne, 55 per cent. of the idiots came from neurotic families.

Dr. Langdon Down,<sup>4</sup> who has made an interesting series of investigations in 2,000 cases, reports 45 per cent. as caused by various neurotic affections in one or both parents. If the mother were afflicted, the first children born were the sufferers; if the father, the later children.

Haller cites the cases of two imbecile women of noble birth and immense wealth who married, and many of whose descendents for more than a century, even to the fifth generation, were idiots.

Esquirol<sup>5</sup> reports the case of an idiot woman at the Saltpêtrière who bore three idiot children.

Dahl<sup>6</sup> made careful comparative studies of the ancestry of 169 idiots and 151 insane. In the former he found 84, or about 50 per cent., had insane relations. In the latter there were 58, or about 38 per cent., whose rela-

<sup>3</sup> Report of the Commissioners on Idiocy to the General Assembly of Connecticut, 1856, p. 35.

<sup>4</sup> "Causes of Idiocy and Imbecility," *Brit. Med. Jour.*, 1873.

<sup>5</sup> Hereditary; Th. Ribot, p. 130.

<sup>6</sup> Bidrig til Kundskab om de Sindssyge i Norge af Ludvig Dahl, Reservenæge ved Gaustad Sindssygeasy, v. 78.

tives also presented marked symptoms of idiocy and insanity. Of these the parents of 18 insane and 21 idiots, about 12 per cent., also exhibited some mental defect.

Of the idiots there were 2 cases where both parents were mentally tainted, 6 where the father, 4 where the mother, 4 where some or all the grandparents, and 5 where the great-grandparents were afflicted with mental disease.

It will be noticed that the great-grandmothers predominated in transmitting the taint, but Dahl gives no explanation of this.

Dr. James R. Dunlop reports the case of a weak-minded father and mother who had seven imbecile children. He was so much interested in the case that he made a number of careful investigations. (*Vide* genealogical table No. I.) For four generations there has been some evident neurotic taint, but it was not marked in the third. The progenitor was a neurotic, and was married to a fairly intelligent woman. To them were born four children, three males and one female. The female was an imbecile and had a son, in no way peculiar, born out of wedlock, but whose father she subsequently married. She had no other issue.

Of the males, one is reported sane, one feeble-minded, and another eccentric if not imbecile. He married a healthy woman eleven years younger than himself. They had eleven children, seven of whom were imbecile or idiotic. One, Jean, has had an illegitimate daughter who was said to be normal. One, Thomas, a hypochondriac, was said to be peculiar in looks and manner, but not weak-minded. He married a sensible woman, and had an imbecile child. James, sane, but peculiar in manner, irritable, and fretful, had eight children, perfectly healthy.

Fletcher Beach also quotes an interesting case where the pre-disposition can be traced through four generations. (*Vide* genealogical table No. II.)

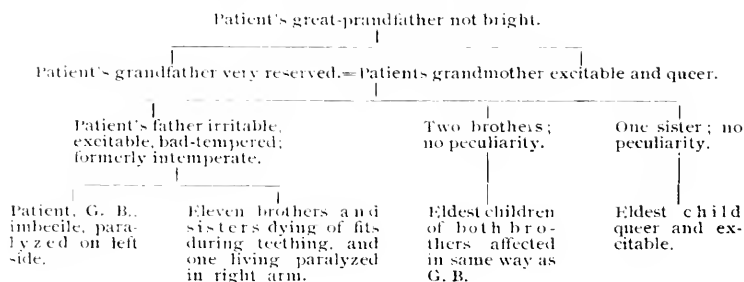
G. B., aged 15 years, an imbecile, paralyzed on left side. Her great-grandfather was not in full possession of his senses. The grandfather was reserved, and the grandmother excitable and peculiar. The father was a neurotic, ill tempered and morose. The father's two brothers and one sister exhibited no peculiarity, but the oldest children of both brothers exhibited the same peculiarity as the patient (G. B.). The sister's oldest child is also peculiar. From the father springs G. B., whose

# GENEALOGICAL TABLE NO. 1.

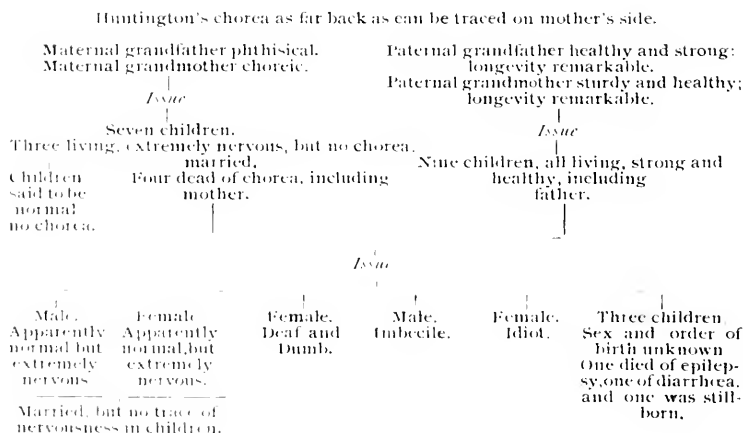
Male, eccentric. Married an active woman, with no obvious peculiarity.

<i>Isaac</i>		Male (John).		Male.							
Female, Ann. Uncommon. Slightly weak- minded; married.	Strange-looking and eccentric; rather weak-minded. Married a sensible, sharp woman, with no tendency, so far as known, to neuroses. He died in 1848, aged 70 years, from "old age and debility." She died in 1860, aged 72, from "disease of liver."	Said to have been weak-minded.									
<i>Isaac</i>											
Male, same	Male, same										
<i>Isaac</i>											
Alexander, Imbecile, born 1800, died 1870, aged 70 years. Cause of death, peritonitis due to congenital hernia	Helen, Same, born 1811, living.	Mary, Imbecile, born 1811, died 1870, aged 59.	William, Idiot, born 1812, living, aged 58.	John, Idiot, born 1814, died 1871, aged 57.	Robert, Imbecile, born 1817, died 1870, aged 53.	Jean, Same, born 1810, died 1863, aged 53.	Thomas, Born 1812, Died 1875, aged 63. Cause of death, chronic bronchitis with nervous exhaustion.	Ann, Imbecile, born 1823, died 1868, aged 45. Cause of death, phthisis.	James, born 1827, living, married.	Charles, Imbecile, born 1832, died 1836, aged 4 years.	
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## GENEOLOGICAL TABLE NO. 2.



## GENEOLOGICAL TABLE, NO. 3.



eleven brothers and sisters died of spasms during dentition. It will be seen by this that the neurosis running through the family touches the third generation but lightly, and intensifies itself in the fourth.

Two family histories of my own patients (*vide* genealogical table No. III.) are interesting studies in heredity:

A. B., apathetic idiot. Huntingdon's chorea has existed in mother's family for many generations. Maternal grandmother choreic, maternal grandfather phthisical. Of this marriage there were born seven children; the order of birth, ages, and sex unknown, with exception of mother of A. B. Three alive and married, and extremely nervous. Have children said to be normal in every respect. The four deceased children, including mother, died of chorea.

Paternal grandfather and grandmother both strong healthy, and lived to unusual old age. Their issue consisted of nine children, sex, age and order of birth unknown. All healthy.

The mother of A. B. was always delicate, and three days after marriage was attacked by chorea, of which she died after twenty-three years. She had eight children, the two oldest—male and female—said to be normal. Both married and have healthy children. Third, female, deaf and dumb; fourth, male, imbecile; fifth, A. B., female, idiot. Three children born after this, of whom nothing is known except that they died in early infancy of epilepsy, diarrhœa, and one was still-born.

H. W., high-grade imbecile, eighth child, first born of twin in a family of fifteen, nine of whom are dead; four from convulsions in infancy, two still-born, one from scarlatina, and two, including patient, of meningitis. Of the six children living, five are said to be normal, and one a shameless harlot, who is practically a paranoiac. Both father and mother came of pronounced neurotic families.

Father peculiar, morose, intemperate, and died in his fifty-first year of apoplexy. His first cousin was an imbecile.

Mother is considered very peculiar.

Howe<sup>7</sup> tells of three towns situated in close proximity to each other in which are a number of families where both parents and children are imbecile. He tells also where an indigent female idiot living in one town mar-

<sup>7</sup> "Causes of Idiocy," p. 78.

ried, with the sanction of the town authorities, a male idiot, who was not a pauper, with three idiot children as a result.

Dr. Isaac N. Kerlin, my master in work, in 1880, presented a valuable paper on the "Causation of Idiocy Based upon the Analysis of One Hundred Idiots and Imbeciles." He found 28 per cent. were caused by insanity and feeble-mindedness, and 57 per cent. by various nervous diseases.

In my own experience based on a careful examination into the family history of 1044 idiots, I find 397 families, or 38 per cent., with a history of insanity or imbecility, and 225, or about 21½ per cent., of various neuroses.

There is one point so nearly allied to our subject as to demand at least a passing consideration.

Consanguinity is commonly accounted a fruitful cause of idiocy, but comparative investigation shows, first, children having both mental and physical defects, the offspring of healthy unrelated parents; second, perfectly developed children with no personal peculiarities whatsoever, the issue of contiguous marriages. This would lead us to accept the statement that consanguinity has but little if any influence in the production of idiocy, unless there be some hereditary neurosis.

Heath maintains that if the blood be pure and uncontaminated there will be no bad results from such marriages.

A confirmation of this may be found in the history of the Hebrews, which gives repeated examples of consanguineous marriages where the intermingling of pure blood gives only good results, and idiocy is the exception rather than the rule.

The statistics that I have been able so far to gather run thus: In 1865 the population of Batz, Brittany, numbered 3,300. Five marriages took place between cousins-german, thirty-one between second cousins, and ten between those of third degree.

The issue of the cousins-german was twenty-three children, free from all disease, both mental and physical. The second cousins had 120 children, normal in every respect, and the issue of the cousins of the third degree were twenty-nine children, also perfect. Two women were sterile. But insanity, idiocy, and nervous diseases were unknown in this community.

Dr. Kerlin found but 7 per cent. of his cases examined directly traceable to consanguinity.

In my own examination of 1,044 idiots, I find but  $3\frac{1}{2}$  per cent. On the other hand, consanguineous marriages where there is the least neurotic taint must always be marked by deterioration of mental power. Naturally, if the taint exist in both parents, the force is but intensified, and idiots are likely to be produced with peculiarities accentuated. A notable example of this is found in Switzerland, where, among the people secluded from the outer world in mountain fastnesses, intermarriage has been going on for centuries. *Here*, by repeated intermarrying, neuroses are preserved intact and idiocy ripens.

Among my own records I find an interesting case which I present herewith:

J. F., excitable idiot, born of cousins-german, in whose families were marked neuroses. Mother, always delicate, and finally died of phthisis. Father, emotional and silly to the verge of imbecility. The following is an account of their offspring: The family, a large one, numbered eleven in all. Four died in early childhood (diseases unknown), three living, of whom I have no history, and four of whom I have records. First born, female, deaf and dumb; second born, female, epileptic; third born, male, idiot; and the eighth born (patient), idiot.

Frequently idiocy appears as the outward and visible sign of the mental deterioration of a family where intermarriage has been frequent, especially one that has been noted for its intellectual qualities, and, according to Griesinger,\* it is a mark of degeneration in a race whose blood has stagnated, as, for example, in the Asylum of L., where many of the proudest names in England are borne by drivelling idiots.

Esquirol says that it is simply impossible to enumerate the idiots in the noble families of France, among whom intermarriage is frequent, and also among the Roman Catholic families of England and Scotland.

We have considered to-night, not possibilities nor chimerical maybes, but grim *facts* gathered through years at much cost and pains, by patient, earnest, thoughtful, philosophic minds. Let us come up from their past with the lamp of experience they have placed in our hands to study the present and verify their conclusions.

What are the signs that tell of this enemy, who shall

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\* Mental Pathology, p. 349.

come in like a flood? What standard shall we lift up against him? It is said that the pulse of a nation's prosperity is found in its merits—the danger signals are read at its source.

Where shall we judge of the intellectual status of a nation but in its educational centres. Schools, colleges, universities multiply with unprecedented rapidity over a land which has just celebrated its fourth birthday among the centuries of civilization.

Through these corridors, out from these doors, press in mad haste for the arena of life, young men and maidens, warm blood, freighted in many cases so fatally with the miasma from some remote ancestor, often doubled and quadrupled in intensity by consanguineous marriages, waiting but for this unhealthy and artificial atmosphere to develop into abnormal growth.

Do we ask why are the hopes of fond parents so often blighted? What becomes of these fair buds of promise?

Go find the answer in those other schools which are growing apace in our midst, dotting from ocean to ocean our own fair land, while others stretch out appealing hands to us—schools public and private which offer no prizes, nor hope of diplomas, yet which are also multiplying with frightful rapidity in response to the agonized cry of the Rachels weeping for their children, and who will not be comforted, because they are not.

The attention of many medical men is drawn to the influence of intemperance in parents as a cause of idiocy. The statistics, however, prove too meagre and the statements too unreliable for us to base any definite conclusions.

The Connecticut commission found 32.34 per cent. My own experience shows 18.38 per cent.

Langdon Down lays special stress upon the intoxicated state of the father at the time of conception, an opinion advanced years ago by Toussenel,<sup>9</sup> and verified in one instance in my own practice.

Ludvig Dahl agrees that to the abuse of brandy by the fathers, and also to some extent by the mothers during pregnancy, may be assigned the most important influence in the production of the large number of idiots in Norway.

Upon the influence of phthisis, held by many as an

<sup>9</sup> "On sait que les enfants se ressentent généralement de l'influence passionnelle qui a présidé à leur conception. La plupart des idiots sont des enfants procréés dans l'ivresse bacchique."—*Monde des Oiseaux*, p. 106. Quoted by Ireland, *Idiocy and Imbecility*, p. 27.



important factor, I have not entered, as it is largely embraced in the points already elaborated.

The last example I have to offer, and by far the most appalling on record, is that of the family known as "The Tribe of Ishmael," whose history, traced through a period of forty years, shows descendants of one unclean, neurotic man, multiplying by consanguineous marriages into two hundred and fifty families, numbering some five thousand individuals, whose continuous criminal record has poured over the northwest a flood of imbecility and crime.

Can we, in face of such evidence, refuse to accept inheritance as one of the fundamental laws of life?

The mark of the beast is ever present—the spoor of the wild animal remains—and to-day we trace it not only to the third and fourth generation, but on *ad finitum*. Not only possible, but the result of inevitable law is this hideous picture :

"That swollen paunch you are doomed to bear,  
Your gluttonous grandsire used to wear ;  
That tongue, at once so light and dull,  
Wagged in your grandma's empty skull ;  
That leering of the sensual eye,  
Your father, when he came to die,  
Left yours alone ; and that cheap flirt,  
Your mother gave you from the dirt  
The simper which she used upon  
So many men ere he was won.

"Your vanity, and greed, and lust,  
Are each your portion from the dust  
Of those that died, and from the tomb  
Made you what you must needs become.  
I do not hold you aught to blame  
For sin at second hand, and shame :  
Evil could but from evil spring."

PROGRESSIVE MUSCULAR ATROPHY, MOST  
MARKED IN THE RHOMBOID AND SHOULDER  
MUSCLES, BUT ALSO INVOLVING THE  
MUSCLES OF THE TONGUE AND FACE, AND  
THE EXTERNAL OCULAR MUSCLES.<sup>1</sup>

BY J. TORRANCE RUGH, M.D., AND CHARLES K. MILLS, M.D.

THIS patient, a married woman, 35 years old, was born in Ireland. Her father died after having been confined to his bed five years with paralysis, the exact nature of which is unknown; her mother is living and well. She has three sisters and one brother living and in good health. She was married when 17 years of age and has three children living. One miscarriage occurred during an attack of typhus fever one year after her marriage. She bore three children in the next three years. Soon afterwards her husband died, and she was obliged to work for her living. She was employed in a laundry and did very heavy work. At this time, about thirteen years ago, the disease from which she is now suffering began.

She was first seized with a severe pain under the right scapula, which lasted several weeks and was pronounced "rheumatic" by her physician. She soon noticed that the scapula was becoming prominent, and for the purpose of correcting the deformity had a corset made extending very high in the back and having firm straps running over the shoulders and fastening in front. This supported the scapula and relieved the pain, but the deformity has gradually increased. For several years she has been unable to dress her hair with her right hand unless she rests the elbow upon some high object. She has had no other attacks of pain about the shoulder until a few days before she was first seen, April 6, 1895, yet felt that she was gradually losing power in the shoulder muscles.

She sleeps well but can only lie flat on her back as any other position causes pain. Her menstruation is regular, but she has dysmenorrhea during the first twen-

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<sup>1</sup> Case presented at the Philadelphia Neurological Society, April, 25, 1895.

ty-four hours of the flow. For the past sixteen years she has frequently had a frontal headache which is aggravated by wet weather. Her appetite is capricious and bowels are irregular.

At first, attention was attracted only to the condition of her shoulders and back. She was unable to elevate the right arm above the horizontal line and elevation of the left was performed with difficulty. She cannot raise the shoulders or move the arm freely forward and backward when these are extended horizontally, nor can she throw the shoulders backward. The peculiar deformities produced when the arms are allowed to hang or



Progressive Muscular Degeneration; Appearance of the Atrophied Muscles of the Back and Shoulders.

when they are held up as far as possible and flexed at the elbows are shown in the photographs. Close examination shows advanced atrophy of the rhomboid, the supra spinatus and infra spinatus, and deltoid muscles on the right side, and the same but to a less degree on the left.

The muscles of the left half of the face are also distinctly atrophied, the mouth and nose being drawn somewhat to the right. The general bulk of the tongue is diminished; and its right half is relatively much more atrophied than the left. Voluntary movements of the tongue are irregular and fibrillary tremors are present.

Involvement of the ocular muscles is evidently be

ginning to take place. In looking both outward and inward the excursion of the left eye is incomplete; and in looking upward it has a tendency to wander outward. Some restriction of movement inward is also observable in the right eye. It is probable that the impairment is of conjugate ocular movement to the left. Pupils respond to light and accommodation.

Touch, pain and thermal senses are preserved. Parts of the body other than those above alluded to, do not seem to be involved in the atrophy.

The case probably belongs to the family of progressive muscular degenerations. It is unusual in having begun in the rhomboid and scapula muscles, and in remaining most marked in them, and for the long period of time elapsing between the two attacks of pain due to the atrophic condition.

**Auto Infectious Anterior Poliomyelitis.** Orcel and Stourme (*La France Med.*, Jan. 4, 1895) report a case of the ascending type of paralysis, of infectious nature, originating in the genito-urinary tract, and which was, for the time being, progressive, but followed by recovery. The patient had a retracted prostate, and underwent internal urethrotomy. Two months later symptoms of general infection followed catheterizations; the urine was scanty and purulent. Then stiffness in the extremities, total sexual impotence, and disturbances of sensibility set in. These disorders spread, and ascended to other parts of the body, causing dyspnoea, muscular atrophy of the leg muscles, decubitus of the sacrum, loss of reflexes, absence of faradic irritability, and bradycardia. Treatment: Mixed anti-specific, without ameliorations; then ergotine, K. I., in small doses, and irrigations of the bladder. Later, complete recovery set in, the symptoms disappearing in reverse order. There was, therefore, first, perineuritis, then myelitis. The authors consider this a case of Duchenne's subacute anterior spinal paralysis.

MACALESTER.

## REPORT OF A CASE OF MELANCHOLIA AT-TONITA.

By THOS. E. BAMFORD, M.D.

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THE following case presents certain symptoms which are recognized and described by Kahlbaum, Séglas, Chaslin, Neisser, Baillarget and others, as katatonia and attonitat, the characteristic of which is that the phenomena of hebetude, or spasmodic and cataleptiform symptoms predominate.

An interesting case is reported by Dr. John Warnock, of Edinburg, in the January number of the *Journal of Mental Science*, entitled "A Case of Catalepsy," in which the symptoms of hebetude and exaltation with excitement were quite evenly divided, and which, in some of its features, closely resembles the case here reported.

L. W., female, age 31, was admitted to this hospital May 23, 1894. He father was insane, as was also an uncle and aunt on the paternal side. She was the mother of four children, and the attack was said to date from her last confinement, which occurred seven months prior to admission. The medical certificate stated that she had been depressed and emotional, and had frequently expressed a desire to take her life. The husband stated that she had gradually become morose and fretful, and on one occasion attempted to shoot herself.

On admission she was reticent and refused to allow a physical examination. She would not talk, walk or make any gestures whatever. When passive movements were attempted, the muscles contracted energetically. This condition was present for about ten days, when she suddenly dropped her torpor, becoming extremely violent and belligerent, attacking the nurses and demanding her release. She soon became exhausted and relapsed into her former condition of inertia. She refused to eat and was mute to all questions. There were no spontaneous movements of the body whatever, and when efforts were made at passive movement they were met

by violent muscular contractions. She was fed twice daily by means of the nasal tube. The respirations were slow and the movements of the chest so slight that they were hardly noticeable—legs flexed on the thighs and the thighs on the abdomen; brows firmly contracted; the lips protruded and were contracted, presenting a pouting appearance. She had incontinence of urine, and the fæces passed involuntary. Three months after admission she suffered from an exhaustive attack of diarrhœa and lost considerably in bodily weight; complete anæsthesia and cyanosis of the extremities became prominent symptoms. Astringents finally checked the frequency of the stools. Although anæmic and much reduced physically, she retained eggs, milk and beef juice in sufficient quantities, and gradually gained in strength. Muscular rigidity became more pronounced. The facial muscles were contracted to such a degree that the face was distorted and presented a frowning, surly expression. After a period of nearly ten months the patient has passed into a condition approaching dementia. When food is placed in her hand she carries it to her mouth. She has a voracious appetite, amounting almost to bulimia: automatic movements of the facial muscles are present. There is slight reaction to the galvanic current. She has gained probably thirty pounds in weight. There is no control over the bladder or rectum and she is absolutely immobile. She has not uttered a word in nearly ten months, nor has she been seen to move voluntarily from one position to another.

The first symptoms noticed in this case were those of depression, lasting about eight months, followed by a brief period of excitement with frenzy, gradually relapsing into stupor and now approaching a condition of dementia. It will be seen that the symptoms in the order of their appearance differ from the description of katatonia in that the symptoms of depression came first instead of the usual stage of excitement, and verbigeration has not been a symptom.

# MERYCISMUS OR RUMINATION, WITH A REPORT OF TWO CASES.<sup>1</sup>

By DAVID RIESMAN, M.D.,

Assistant Demonstrator of Pathological Histology, University of Pennsylvania  
Instructor in Clinical Diagnosis, Philadelphia Polyclinic; Attending Physician for Nervous Diseases, Northern Dispensary.

**A**MONG the aberrations of function presented by the human stomach, none is more interesting or more strange than that known as rumination or merycismus (*merukismos*, ruminaton).

Although the ancient writers, particularly Aristotle, were acquainted with rumination in animals, they have left no records indicating that they possessed any knowledge of its occurrence in man. The first author to mention it was the great Italian anatomist, Fabricius ab Aquapendente,<sup>2</sup> the teacher of William Harvey, who lived at Padua towards the close of the sixteenth and the commencement of the seventeenth century. In one of the chapters of his book, entitled, "*De Ruminacione*,"<sup>3</sup> he describes with much clearness two cases of merycismus—the one was a nobleman who is said to have had two horns on his forehead; the second a Paduan monk who ruminated because his father had a little horn on the forehead—"quod ejus pater paulo supra frontem in capite corniculum durissimum."

Since Fabricius' time quite a large number of cases have been reported, particularly by European writers, but only a few instances have been observed in this country. The literature has been carefully reviewed by Johannessen<sup>4</sup> and by Singer,<sup>5</sup> to whose monographs the

<sup>1</sup> Read before the Philadelphia Neurological Society, March, 1895.

<sup>2</sup> Fabricius ab Aquapendente, Hieronymus. *Opera omnia anatomica et Physiologica cum Prefatione*. J. Bohmii, Lipsiæ, 1687, p. 135.

<sup>3</sup> Fabricius gives the following excellent definition of rumination: "Ruminatio igitur nihil aliud est, quam cibi in os regestio-vel clarus, ruminatio est localis cibi motus quo sursum per gulam in os regeritur, manditur, et laevigatur, inde rursus devoratur." "*De Ruminacione*," p. 135.

<sup>4</sup> Johannessen. *Zeitsch. f. Klin. Medicin.*, Bd. x., p. 264, et seq.

<sup>5</sup> Singer. *Deutsch. Archiv. f. Klin. Medicin.*, Bd. li., 1893, p. 472, seq.

writer would refer for a more extended bibliography than the one given at the end of this paper.

We may define rumination as a periodic regurgitation of the food unattended by nausea, retching, or disgust, the regurgitated material being either voluntarily ejected from the mouth or again swallowed, remastication not being an essential part of the act.

Writers are generally in accord in considering this regurgitation analogous to the process of rumination in the lower animals. In them we find that rumination is peculiar to certain classes of herbivora, being evidently a developmental or evolved character, the result of adaptation to environment. Herbivora, particularly the ruminating genera, in the wild state are compelled to ingest large quantities of coarse food very hastily, because, to use the words of Robert Meade Smith,<sup>6</sup> "in eluding their enemies, they rely chiefly upon sharpness of vision, acuteness of hearing and agility." They have in consequence come to be provided with a peculiarly complicated and capacious stomach, which in the majority of ruminants consists of four compartments: the rumen or paunch, the honey-comb bag or reticulum, the psalter, omasum, or maniplies, and the abomasum or rennet. Of these the last is alone comparable to the human stomach.

The food after its imperfect primary mastication, passes into the first and second stomachs, while fluid and finely divided particles may at once enter all four compartments. Rumination begins a variable time after the meal. The bolus is formed and regurgitated into the mouth, remasticated, and then swallowed in a state of fine subdivision, permitting it to pass through the narrow aperture between the reticulum and maniplies. From the latter it is transmitted to the true stomach to be there subjected to the action of the proper digestive juices.

The factors bringing about this complicated process are principally two—a lessening of the intra-thoracic pressure and an aspiration of the gastric contents.

At the moment at which regurgitation begins the glottis is closed and the diaphragm is forcibly contracted; this leads to an enlargement of the thoracic cavity and a consequent rarefaction of the air. The decrease in pressure causes, on the one hand, an aspiration of the blood in the jugular veins into the heart; on the other, a suc-

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<sup>6</sup> Robert Meade Smith. *Physiology of the Domestic Animals*.



tion of the gastric contents into the esophagus, the cardiac orifice being synchronously relaxed. As soon as the bolus has entered the gullet, it is carried upward by an antiperistaltic movement.

The stomach itself is probably passive, as we may infer from Magendie's famous experiment on the mechanism of vomiting. Magendie found that when he excised the stomach of a dog and substituted for it a bladder, the injection of tartar emetic was just as capable of producing vomiting as before. Regarding the participation of the abdominal pressure, writers are not in complete unison, some holding that it is necessary, others that it is not.

Rumination is a reflex act and is presided over by a centre situated in the medulla oblongata, the afferent pathway being constituted by the pneumogastric nerves, the efferent by the phrenic nerves and the motor nerves of the abdominal muscles, the stomach, and the esophagus.

As far as the process of rumination has been studied in man, it has been found identical in its mechanism with that of herbivora, the factors of paramount importance being the closure of the glottis and the descent of the diaphragm. But while the mode of its production has been quite fully elucidated, we are as yet ignorant of its ultimate cause.

The anatomic lesions that have been noted have thrown no light on the origin of the phenomenon.

Arnold<sup>7</sup> and Luschka<sup>8</sup> found in their respective cases dilatation of the lower end of the esophagus, the so-called *antrum cardiacum*, and also observed an unusual thickness of the internal branch of the spinal accessory nerve: that which unites with vagus. As in ruminating animals the gullet terminates in a funnel-shaped expansion (the *gouttière esophagienne* of the French) and as in them the inner branch of the accessory nerve is very thick, Arnold and Luschka concluded that the existence of these anomalies accounted for rumination in man.

But unfortunately for this theory the cardiac antrum has been found in cases that did not present merycism during life, while in other instances the esophagus was normal: and as regards the hypertrophy of the nerve,

<sup>7</sup> Arnold. *Untersuchungen im Gebiete der Anatomie und Physiologie*, Bd. i., p. 211, Zürich, 1838. Quoted by Singer.

<sup>8</sup> Luschka. *Virchow's Archiv.*, Bd. xi., 1857, p. 247. Quoted by Singer.

its size is known to vary considerably under normal conditions.

In Singer's cases Von Hacker was able to determine, by esophagoscopic examination made *intra vitam*, a probable dilatation of the lower end of the esophagus, which Singer attributes to the patients' habit of swallowing imperfectly masticated pieces of food. In a few cases the stomach has been found dilated.

Bourneville and Ségla<sup>9</sup>, in two autopsies, found both stomach and esophagus normal.

All these contradictory observations prove that there is no constant anatomic substratum, whence it follows that we must rank the condition, at least for the time being, among the functional disorders of the stomach, and consider it, as Oser<sup>10</sup> has done, a motor neurosis.

As both experimental examination and esophagoscopy have shown that the cardia is invariably closed toward the stomach, except during the transit of the food, we must of necessity believe that in rumination there is, as in vomiting, a temporary relaxation of the orifice. That a permanent insufficiency exists, as has been claimed, is unlikely, as this would inevitably lead to a perpetual regurgitation of everything ingested.

An important element in the causation of rumination is unquestionably the neurotic constitution. Nearly all writers are agreed on that point.

A German physician, Näcke,<sup>11</sup> who is a sufferer from the affection, and who has studied his case minutely, is convinced of its relation to neurasthenia. Whenever his nervous system is most unstable, the rumination is most energetic.

The frequency of the disease in the educated—physicians, students, and clerks—would also indicate its alliance to the neurasthenic state.

In a few instances, notably in the case reported by Körner,<sup>12</sup> in which a governess taught the practice to her two wards, the habit was acquired by imitation.

Bourneville and Ségla, as well as others, have pointed out its occurrence in idiots and the insane. In such cases it may be dependent upon bulimia.

<sup>9</sup> Bourneville et Ségla. *Du Merycisme*. Quoted by Singer.

<sup>10</sup> Oser. *Die Neurosen des Magens und ihre Behandlung*. Wien, 1885.

<sup>11</sup> Näcke. *Neurolog. Centralblatt*, 1893, xiii., p. 2.

<sup>12</sup> Körner. *Archiv f. Klin. Medicin*, Bd. xxxiii. Quoted by Singer.

The disease is far more common in males than in females; as a rule it causes no serious disturbance unless the patient habitually rejects the regurgitated food. With but few exceptions it seems to have resisted every form of treatment. The phenomenon is not generally under the control of the will.

Something ought to be said concerning the condition of the gastric juice;—here again we meet with the same variability as in the morbid lesions, perhaps in the majority there is subacidity, in some even achlorhydria, as in the two cases here reported, although a sufficient number of analyses was not made in them to predicate positively the absence of hydrochloric acid. In a few instances, as in the case of Alt,<sup>13</sup> hyperacidity was noted. It was shown by Linossier and Lemoine,<sup>14</sup> that the acidity varies in the same case on different examinations.

The symptomatology is pretty constant. The writer cannot better describe it than by detailing the histories of two cases that he has recently observed.

Case I. Mr. Wm. S.<sup>15</sup> was sent for a physical examination by Dr. Stem, of Berlin, N. J. He is 44 years of age, married, an Englishman by birth, an engineer by occupation.

*Family history.*—Father died of asthma, mother of consumption. There is no neurosis in the family, no one suffers, as far as he knows, from stomach troubles.

*Previous history.*—At the age of eighteen he had white swelling of the left knee; suppuration ensued and continued for many years; after a period of arrest, the sinus opened again and continues to discharge up to the present time.

He came to this country at the age of sixteen years, has always worked faithfully at his trade, was healthy, not intemperate in the use of liquor or tobacco, nor guilty of sexual excesses. He has been married twice, and is the father of twelve children, of whom eight are living and well. During the last three years he has had a number of pulmonary hemorrhages; has had cough and has lost flesh; night sweats have not occurred. He has always been a large eater, very fond of meat, but does not think that he eats inordinately fast, and states that he chews his food fairly well.

<sup>13</sup> Alt. *Berliner Klin. Woch.*, 1888, pp. 519, 544.

<sup>14</sup> Lemoine et Linossier. *Compt. rend. Soc. de la Biol.* 1893, 98., v. p. 339.

<sup>15</sup> This patient was shown before the Neurological Society.

*Status presens.*—The patient is below average height, pale, and somewhat emaciated, and weighs 115 pounds. There is partial consolidation of the right lung; expiration is prolonged and bronchial, particularly toward the manubrium sterni. Fine subcrepitant rales are audible over the infiltrated area. The heart sounds are normal except for accentuation of both second sounds at the base. The liver dullness extends in the mid-clavicular line, from the sixth rib to the costal margin; the spleen is not enlarged.

There is no epigastric tenderness or prominence.

The stomach is apparently not dilated; outlined by means of auscultatory percussion, the long diameter is found to be  $16\frac{1}{2}$  cm., the depth  $8\frac{1}{2}$  cm. Of the deglutition murmurs, only the first one, that synchronous with swallowing is audible.

There is lateral spinal curvature; the back is dotted over with small areas of atrophy of the natural pigment.

Tubercle bacilli are present in the sputum. Examination of the blood gave 4,975,000 red corpuscles, 12,500 white corpuscles, and 82% hemoglobin. The fact that the count was made about an hour and a half after a large meal may explain the slight leukocytosis.

The urine is normal.

The habit of ruminating, the existence of which was discovered on investigating the patient's history, first manifested itself about twenty years ago; he cannot recall the circumstances connected with its onset, but is positive that at the time he was in perfect health, regular in his habits, not dyspeptic, nor had he seen anyone subject to the same affection.

From that day until the present writing he has, with occasional intervals of short duration, presented the following condition: About half an hour after meals the food begins to return to the mouth without the slightest effort and without the faintest intimation of nausea, and possessing its natural taste. He has no difficulty in distinguishing the kinds of food—if he had eaten of a large number of articles, he could readily differentiate among them. The quantity regurgitated is small and fills the mouth comfortably. If he is out in the open, he generally rejects the food; but if indoors, or in the cars, or busily engaged, he swallows it again. Regurgitation is most apt to occur after the noon-day meal.

As a rule the material is finely enough divided not to require a second chewing, but at times it contains

pieces of meat of considerable size, these he picks out and remasticates before swallowing.

The whole process repeats itself at intervals of from five to ten minutes, for about one and a half to two hours; when he is sitting quietly the intervals are somewhat longer. At the end of rumination the food tastes just as pleasantly as at the beginning.

At times, however, the stomach contents become sour; when such a condition exists, the regurgitation continues for about three hours, the material constantly growing more acid until it tastes like vinegar and nearly "cuts his throat" as he expresses it.

He can neither suppress nor inaugurate the process of regurgitation directly, but by assuming the recumbent position he can check it unless he has eaten certain articles, like rice-pudding or smoked meats.

The order in which the food returns depends less upon the order in which it is ingested than upon its nature. Rice-pudding, he thinks, whether eaten first or last, would always come up first. The particular articles which are most liable to be regurgitated are, rice-pudding, smoked sausage, smoked fish, especially mackerel; eggs, ham, pie, and other sweetmeats; at times bread and butter.

Among liquids, he names porter and beer. Beer, he states, comes back as frothy and refreshing as when he drank it; indeed, he thinks, he enjoys it more the second time; whisky does not return; coffee does; tea does not; milk is regurgitated uncurdled for half an hour after ingestion.

Rumination is always preceded by a feeling of fullness in the epigastrium; but there is no belching, although eructations are common between meals, after regurgitation has ceased. To the patient it seems as if the food was forced up by gas collecting beneath it and propelling it upward. He is conscious of its entire passage from the stomach along the gullet to the mouth.

Ordinarily, the ingesta return perfectly sweet continuously for days and weeks; he is then entirely comfortable. But at intervals, for unaccountable reasons, the food becomes acid, and after a few days severe, cramp-like pains develop on each side of the epigastrium. As soon as the bowels are freely moved, the pains cease and the acidity of the food disappears.

Occasionally, when the acidity reaches an extreme

degree, the bowels will become loose spontaneously, the acid material apparently acting as a cathartic.

He has never vomited. When he was asked to what he could compare his symptom, he said it resembled more the chewing of the cud by the cow than anything else.

The regurgitated material is small in amount, semi-liquid, and of a mush-like consistence. The writer had the opportunity of examining it twice, on different days.

It was acid in reaction, and of a sour but not offensive odor. Uffelmann's test indicated a trace of lactic acid; the presence of HCl could not be demonstrated either with Congo-red paper or Günzburg's test. One of the specimens had separated into three distinct layers—the upper and lower consisting of flocculi of food material, the middle of a turbid fluid holding small particles in suspension. The first sample contained two pieces of meat,  $1\frac{1}{4} \times \frac{1}{4}$  and  $\frac{3}{4} \times \frac{1}{4}$  inches respectively.

On one occasion, the gastric contents were removed with the stomach tube, two and one half hours after an unrestricted meal. About 100 c.c. of a sour-smelling liquid containing solid particles were obtained.

The reaction was acid to litmus. HCl was absent, lactic acid was present in considerable amount. The total acidity, which was, of course, not due to HCl, corresponded to 3.4 c.c. of a deci-normal sodium hydrate solution. Neither starch nor erythrodextrin were present; but with Fehling's test a large quantity of sugar was found; a trace of albumin was also present; the presence of propeptone could not be positively determined; there may have been a trace; the biuret test was readily obtained, even after the filtered fluid had been boiled, indicating the presence of peptones.

On boiling the fluid exhaled a most delightful aromatic odor, difficult to describe.

Dr. Stem has treated the patient in various ways; latterly under the use of a carminative mixture containing *Oleum caryophylli*, *Sp. chloroformi*, *Tr. capsici*, *Tr. nucis vomicae*, and *Tr. cardamomi comp.*, the rumination has ceased. During the last three weeks he has regurgitated the food but once. Yet, strange to say, he feels far more distress in his stomach now than when the rumination was active.

CASE II.—R. B., *æt.* about 60; married; Pennsylvanian by birth; contracting carpenter.

Family history is negative except for the fact that a

niece had for a number of years the same trouble from which he suffers. No history of neuroses. Previous history.—The patient had typhoid fever and articular rheumatism in his youth.

He was always a large and rapid eater, his favorite dish being to the present day salt pork. In the fall of 1893 he had influenza from which he recovered rather slowly. He now has tuberculosis of the right lung; the sputum contains tubercle bacilli; the stomach is not dilated; the outline obtained on auscultatory percussion measures 19 cm. transversely, and 10 cm. in depth. Three deglutition murmurs are audible: the first immediately on swallowing; the second in seven, and the third in twenty seconds.

From his early boyhood, perhaps from the age of twelve, until he was twenty-two years old, he was addicted to rumination. He has no idea whatever of the cause of the habit; he was, as has been stated, a large meat eater, and was inclined to bolt the food.

Being in the habit of discarding the regurgitated food, he became greatly reduced in strength, and was treated for a long time by an able country practitioner for dyspepsia, but without effect. Finally, a few doses of a medicine given to him by an herb doctor cured the complaint. He gave the same remedy to his niece with a like result.

Two years ago the condition returned and has persisted until the present day. Rumination begins about five minutes after the ingestion of food and continues for nearly three hours. Generally there are, he thinks, about six distinct acts of regurgitation. There is neither nausea nor vomiting, and the food tastes as sweetly as in the beginning. As a rule he ejects the bolus, but at times swallows it; large pieces are chewed a second time. He has no control over the process, nor can he at will institute it; he has more distress in the stomach when he ruminates than when he does not.

During active out door exercise, the habit is generally in abeyance; inactivity or the dorsal decubitus intensifies it. In this particular he differs from Case I., in whom the phenomenon is most energetic during exercise and least so during repose. The regurgitation is preceded, as in Case I., by a feeling of wind in the stomach.

Among the articles of diet ice-cream, bread, pie, apples, rice-pudding, milk, and especially fat meat are

most prone to return. He told the writer that if he ate twenty different dishes, he could distinguish them all in the ruminated matter.

Two small specimens of the food were obtained for examination, one regurgitated one half hour, the other two hours after dinner. Both were sour and semi-solid, the second being somewhat more liquid than the first.

The reaction was acid, HCl was absent, lactic acid was present only in traces, if at all.

On account of the tuberculosis the patient was placed on creasote, with the result that both the pulmonary symptoms and the gastric condition improved, but a complete cure of the rumination has not been achieved.

These two cases, the histories of which have been given, correspond in many particulars, but very strikingly in the fact that the stomach condition is not associated with a general neurasthenic state, as was the rule in a large number of cases reported in literature.

There may be a relaxation of the cardia, but the writer believes this to be a favoring or predisposing, not an etiologic factor. The coexistence of phthisis with the *mercyismus* is merely an accident.

We have in both patients a history of a most too liberal indulgence in meat diet, linked, clearly in Case II. and probably in Case I., with imperfect mastication. The gastric juice in both is deficient in HCl. The first is a common failing, yet rumination is rare; the second may rank among the causal factors of the *mercyismus*, but *achlohydria* is not constant; in some cases there has been hyperacidity, it is possible, however, that different conditions of the digestive juices may lead to the same phenomenon, and the writer is inclined to attach considerable importance to the subacidity and consequent fermentation, particularly since both patients ascribe the inauguration of each act of rumination to a distention of the stomach with gas.

The theory of atavism which explains a number of obscure functional and structural anomalies can scarcely be applied to the condition under consideration, as the connection between man and the ruminating herbivora, if there be any, is too remote.

In addition to the two cases which have come under the writer's personal observation, he has heard of two others that presented the symptom at one time but are now free from it. If these two cases be taken into account, we have four instances of rumination, a compara-



tively large number, which justifies the belief that the condition is more common than the references to it in text-books would seem to indicate. The patient is apt to consider his affection a peculiar form of vomiting, and not until he is carefully questioned, will its true nature be revealed.

The treatment of merycismus is exceedingly unsatisfactory, very few instances of cure being recorded. The carminatives, electricity, nerve sedatives, and other agents have been employed, but as a rule with but temporary results. In those cases in which the gastric juice is greatly altered, a correction of the defect may bring about a cure. Thus in Alt's case, in which hyperacidity existed, treatment directed against that state, permanently stopped the rumination.

In view of the usual underlying condition, neurasthenia, something might be hoped for from an improvement in the patient's general state.

**Dr. Robert Chase** and Dr. John Chapin, at a meeting of the Phil. Co. Society, presented the subject of "Heredity in Insanity and in Idiocy." (*Medical News*, March 6, 1895.) Dr. Chase presented the general laws which heredity follows, and Dr. Chapin in his paper formulates the conclusion as follows:

"1. Physical characteristics, and those distinguishing the human species are transmissible as an inheritance.

"2. Knowledge, genius and culture are not an inheritance, but depend rather on influence, education and environment. Mental receptivity is transmissible; psychic qualities are not necessarily an inheritance, requiring favorable surroundings and circumstances for growth and development.

"3. Insanity as a disease is not transmissible by inheritance, but may be acquired or evolved from a neurotic heredity as a basis.

"4. A neurotic predisposition is transmissible by inheritance, but there is no absolute rule that it will be transmissible in any given case or in any case.

"5. Ingrading of neurotic temperaments is most conducive to the creation of a neurotic heredity.

"6. Idiocy and imbecility may be a defect, having an origin in consanguineous marriages, parental conditions, accidents, arrested developments, infantile meningitis, tuberculosis, and a lack of potency on the part of one of the parents from unexplainable causes." PHELPS.

## Asylum Notes.

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### *Among the Hospital Reports.*

Appended to the written report of each hospital for the insane are the statistical reports of clinical study. Unfortunately they are for the most part without any explanatory comment to make them clear.

As the report as a whole may not be wholly representative of the daily internal activity of the hospital, yet they are the most accurate indications we have, and as such, issued for the purposes of comparison, we would note some of the most important central topics there found.

*Congregate Dining-rooms.*—Custodial and building devices prevail in the reports. Under building ideas of custodial character, congregate dining-rooms or dining-rooms for congregating large numbers of the insane from diverse wards at meal time, have formed during the last decade a prominent idea. It is a live question at present. Dr. Rogers (Longcliff, Ind.) has for a year or more had a congregate dining-room, and reports in favor of them, although not for over 50 per cent. of his patients. Dr. Tobey (Toledo, Ohio,) has advocated them most enthusiastically. Dr. White (Milwaukee) has also favored them very much.

On the other hand, Dr. Ratcliff (Dayton, Ohio,) says, "In his judgment they fail to accomplish the purpose desired." Dr. Burgess (Montreal) speaks very decidedly "that from twenty years of experience with both systems of dining-rooms, I infinitely prefer the separate." Dr. Russell (Hamilton, Ont.) is not only emphatic, but shows the more powerful argument, in that he has been to the trouble of changing back to separate dining-rooms.

Briefly and fairly stated, the various arguments found in reports at various times are about as follows :

1. It costs less to build one large dining-room than many small ones. This is economy in "first cost."
2. Economy in "scraps" or "waste" is said to be greater in one large dining-room.
3. The physician can supervise the diet better.
4. Meals can be put on warmer and with fewer waiters.
5. In a vague way it is claimed to be good for the patients to get out three times daily in their trips to and from the dining-room; seemingly by reason of the change and ventilation.

*Contra Arguments.*—1. The cost in original saving of one large dining-room over that of several small ones, is not yet proven by figures.

The method of figuring by the saving of beds is rather fallacious. If a congregate dining-room for 500 be estimated at \$15,000, then an equal amount of room on the wards, estimated by proportional costs, and of the same construction, would probably cost not quite so much.

2. Economy in "scraps" is not enough to have much weight.

3. The greater supervision is a valid argument, but of not enough value to outweigh any disadvantages.

4. The only saving of warmth to meals is by having the kitchen near at hand (say two to four minutes).

5. The grouping of people of diverse habits, neatness, violence, and tastes is offensive and directly against the whole modern idea of individualization.

6. The trip for the people up and downstairs, the outdoor exposure in all weather, the waiting to congregate and form in line, is a great disadvantage and very unhomelike.

7. It leads one to classify according to dining-room attendance, which interferes with other essentials in classification.

*Cottage System.*—The next most important element discussed is the cottage system, or the building of detached cottages instead of one large building.

This construction has been a popular and prevailing ideal for the past ten years. Kankakee, Toledo, Athens, Jamestown and others have been leading. The arguments in favor of cottages are greater subdivision and classification of patients, and a more homelike form of living.

Even in pursuance of these praiseworthy ideas, how-

ever, it would seem to be the fact, that the separation can be as complete, and the classification fully as perfect in form by adjustment upon wards, as by adjustment in separate buildings. The expense and difficulty of heating, the difficulties of supervision, of feeding and furnishing, are the objections to the cottage system. By having short pavilions between the wards and buildings, and by having them somewhat lower and more spread out, it would seem possible to secure the desired subdivision as well.

It will always be cheaper and less difficult to supervise one building than a small village of cottages. The best mode of building is still to be considered an open, debatable question, and one which should be discussed very thoroughly by those intending building.

*Medical Work.*—Progress is being made, as shown by various reports.

And if only “progress” be the watchword, high attainment is secured. The best worded statement, perhaps, is found in the report of Clarke Gapen, the new superintendent of Kankakee, Ills. He says:

“The medical and nursing staff have undergone many changes, but throughout have maintained a high average of proficiency. During the past year, especial effort has been made by the medical staff, to qualify itself for its high and difficult work, by courses of study in histology and pathology of the brain, under the direction of Dr. Meyer, and in close analytic study of diseases of the mind in general, and especially of the cases coming under treatment.

“A determined effort has been made on the part of the medical staff, not only to know the newest and best that science in any part of the world had to offer, but to do the best that the art of medicine could invent for the amelioration of bodily and mental afflictions with which we are surrounded.

“Rarely, I believe, in the history of American hospitals, has a more united and spirited effort been made to obtain high scientific and philanthropic results. Daily meetings of the medical staff have been held to report to the superintendent the conditions prevailing in all departments of this great hospital, to consult together how best to obtain desired results, and together examine and study critically the more complicated and difficult cases. I can be all the more unstinted in my praise, for the reason that my own energies have

been largely absorbed in economic and constructive reform, and in my relations to this part of the work, mainly that of general direction."

P. M. Wise (Hudson River Hospital, N. Y.,) outlines also similar revivalistic measures of the past two years, and gives papers written by the staff, each one of whom investigated along some special line. He had a daily consultation hour and kept up the training school. Such reports as these seem the result of a common sense study, made from an inside position, and are, therefore, probably the best answers made yet to Dr. Mitchell's criticisms. In these reports, moreover, the element of the assistant physician is made prominent. It, as they themselves claim, the superintendent must be mainly a business man, then the one having charge of the nursing, the medical and the scientific work, ought to rank up, so that able men be secured. The general administrative ability must be most able as a matter of course, but the other, the medical and scientific, seems to be the ones most liable to be neglected in our State hospitals.

Catonsville, Md., and Tuscaloosa, Ala., have not the clinical reports of preceding years. The Government Hospital and Norristown Hospital have pathological reports, though not made by the staff.

The Michigan reports (of Pontiac and Traverse City) review the medical work in an instructive way. Indeed, a review of the work which the report covers, with comments, would seem to be the more logical function of a report than would be the publishing of essays.

Middletown Hospital, N. Y., has an essay attempting to prove the correlation of physical and psychical abnormalities. This is the result of a three months' study by a physician not on the regular staff. Long lists of measurements are given, among which we may mention "irregular ears," "irregular ears immoderately," "flat ears," "flat ears immoderately," "face masculine," "feminine," "asymmetrical," "nose situated too low," "depression on occipital fontanelle," "depression over one or more sutures," "occiput irregular," etc., etc. One thousand insane are compared with 110 sane people.

Many other hospitals doubtless are doing extra routine work which is not noted in the reports. Indeed, we know of over a dozen. The spirit of research and competitive work is growing.

*General Paresis.*—Among the clinical tables so variable is the personal equation, that we cannot select many

topics. Perhaps concerning general paresis we can learn something, as it is the most thoroughly definite disease entity in the classification tables.

Hospitals report variously. We note the following as being the most frequent and least frequent of the proportion of paretics to admissions as given in the reports so far received:

Of those reporting more than one paretic to twenty admissions, we have:

Pontiac,	-	-	-	1 to 8.3.
Danvers, Mass.,	-	-	-	1 to 8.5.
Westboro, Mass.,	-	-	-	1 to 11.
Traverse City, Mich.,	-	-	-	1 to 14.
Taunton, Mass.,	-	-	-	1 to 14.
Rochester, Minn.,	-	-	-	1 to 15.
Kalamazoo, Mich.,	-	-	-	1 to 17.

Of those reporting less than one paretic to one hundred admissions, we have:

Austin,	-	-	-	0 to 126.
Warren, Pa.,	-	-	-	1 to 250.
Catonsville, Md.,	-	-	-	1 to 109.
Mt. Hope, Md.,	-	-	-	1 to 160.
Norfolk Neb.,	-	-	-	1 to 130.
Petersburg, Va.,	-	-	-	0 to 176.

The above shows evidently no distribution by city population, or by section of the country and, in fact, gives no clue based upon ordinary causes. Of course, the personal equation of the physician making the judgment has some effect, as well as the fact that some cases not diagnosed on admission as paretics, are so designated later. A judgment based upon the number of deaths would be more accurate, if only paretics were always noted in those cases. Still, there is doubtless a capricious variability about the disease not yet fully studied.

As to causation, although one can always hold a suspicion of syphilis, yet is only proven in a minority of cases. [List of Reports not yet complete.]

## Periscope.

### PHYSIOLOGICAL.

**Voluntary Motion as Influenced by Sensory Disturbances.**—D. T. Polakoff (*Archive of Psychiatry, Neurology and Legal Psycho-pathology*, Russian).

P. comes to the following conclusions reached by aid of the experimental method :

1. The anaesthesia of the upper lip of the horse as produced by bilateral section of the infra-orbital nerves effects a paralysis like state of the upper lip (pseudo paralysis), so that voluntary movements of the upper lip with the purpose of seizing the food are, impossible ; to compensate this defect the animal uses its lower lip, tongue and teeth with increased energy. This paralysis is not complete as occasionally voluntary movements of the upper lip with the purpose of seizing the food are noticed, but these movements are rare, weak and inco-ordinated.

Unilateral section of the infra orbital nerve of the horse causes slight although distinct disturbances of the voluntary movements of the upper lip.

2. Anaesthesia of the tongue of dogs is effected by bilateral section of the lingualis, and section of glosso pharyngeus nerves causes slight disturbances of the swallowing movements of the tongue, which disturbances soon disappear entirely. But if the mucous membranes of the hard palate, gums, mouth and cheeks of the dogs thus operated upon, or of normal dogs (to the latter the tongue must also be cocaineized), are made anaesthetic by pointing with a five per cent. watery solution of cocaine muriate, the swallowing movements of the tongue become intensely disturbed ; they become weak, awkward, inco-ordinated, and do not fulfill the purpose they are intended to serve.

3. The above mentioned motor disturbances of the upper lip of the horse and of the tongue of the dog depend exclusively on the anaesthesia of these organs, as a lesion of motor paths peripheric and central was out of question.

4. Intense disturbances of the voluntary movements of the anaesthetised organ will follow only when the anaesthesia is complete and involves the whole organ, or eventually even the adjoining parts ; the complete anaesthesia of the tongue of dogs did not suffice to effect marked disturbances of its voluntary motions, this result being reached, however, when besides the tongue itself, the mucous membranes of the mouth, gum and cheeks were made anaesthetic.

The author concludes that for the primary excitation and appropriate performance of voluntary movements, simultaneous or previous sensory impressions, are needed. Under normal circumstances the sensory stimulus comes from the given organ itself, and communicates itself in the most direct way to the corresponding subcortical or cortico motor centre. When the said organ is anaesthetic the sensory impulse has to come from adjoining, or eventually even from removed organs in which case it will have to pass through unusual paths to the motor centres of the given organ, but may yet be a sufficient impulse for the production

of appropriate voluntary movements. Thus it is explained how patients with extended anesthesia of extremities may walk well, press one's hand or hold some object in their hands, if they perform these actions with open eyes, that is by help of visual impressions, while with the eyes closed their gait becomes disturbed, they fall easily, they let the object fall from their hands, etc. ONUF.

#### PATHOLOGICAL.

**Crossed Hemianesthesia and Hemiparaplegia (Syndrome de Brown-Séquard) From Medullary Concussion.**—Reynes (*Gazette des Hôpitaux*, March 26, 1895), from a study of this subject comes to the conclusions that—1. Medullary concussion by causing unilateral lesions (hematomyelia?) can give rise to the syndrome of Brown-Séquard. 2. Medullary concussion can cause a dilatation of the iris. 3. The points of origin of the great sympathetic are in the dorso-lumbar medulla. FREEMAN.

#### CLINICAL.

**The Differential Diagnosis of Traumatic Intracranial Lesions.**—Charles Phelps (*N. Y. Med. Journal*, November 10, December 8, 15, 22, 29, 1894, and January 5 and 12, 1895).

After an exhaustive and critical consideration of the above subject from a practical point of view, the author summarizes his conclusions as follows:

**Hæmorrhages.**—The morbid conditions which may directly result from traumatic intracranial hæmorrhages are: an abnormal temperature, a complete or partial loss of consciousness, a change in the character or frequency of the pulse or respiration, a disturbance or abrogation of muscular function, and an irregularity of the pupils. These conditions are subject to complication, modification, or supersedure by the symptoms of coexistent lesions.

A continued subnormal temperature is characteristic of large and comparatively uncomplicated hæmorrhages, and as these are more frequently of epidural character, it may be regarded as to a certain extent diagnostic of the variety as well as of the class. The absence of symptoms indicative of parenchymatous injury will be confirmatory of the opinion that an existent hæmorrhage is derived from the epidural vessels. Associated symptoms of diffused contusion suggest a pial, and those of laceration a cortical, hæmorrhage. In the majority of cases the primary record of temperature is from  $99^{\circ}$  to  $99^{\circ}+$ , and in any case in which, then or afterward, it exceeds  $101^{\circ}+$ , or probably  $100^{\circ}$ , the elevation is due to an associated lesion. It follows that in pial or cortical hæmorrhages the temperature has a higher range than in those of epidural origin, and is proportionate to the extent and importance of the complication. The bilateral variation to which the axillary temperatures are subject is not peculiar to this result of injury.

The primary unconsciousness which is of frequent occurrence in cases of hæmorrhage is a symptom of complicating general contusion; the secondary unconsciousness, due to the loss as well as the pressure of blood effused, follows with or without an interval of restored consciousness, dependent upon the severity of the diffused injury of the parenchyma and the rapidity of the hæmorrhagic effusion, and is partial or complete in proportion to its amount. Consciousness is always lost in fatal cases; it is retained in fifty per cent. and more in recovering cases, even in those demanding operation.

The character and frequency of the pulse have no definite relation to the form, location, or amount of hæmorrhage. The pulse may be normal, slow, or frequent in large extravasations wherever situated; but frequency is of so much more usual occurrence in hæmorrhage than in



other intracranial lesions that when noted it may be considered fairly diagnostic, with the numerical probabilities in favor of its epidural character. The bilateral variation in the force and fullness of the arterial pulsations is common to hæmorrhages and to injuries of the brain substance, and of importance, therefore, only in general diagnosis.

An alteration in the character or frequency of respiration is almost invariable in fatal cases in which hæmorrhage is an approximately isolated lesion. When the effusion is upon the convex surface of the brain, respiration is usually frequent and often stertorous; when at the base posteriorly, it may be frequent with cyanosis, or, if pressure is made upon the medulla, it becomes progressively slower until it ceases altogether, though cardiac and arterial pulsation may still continue. In recovering cases it is habitually unchanged. In complicated or mixed cases it, like the pulse, perhaps as a resultant of opposing forces, very generally remains normal; and if abnormal it is more likely to be stertorous than unduly slow or frequent.

General or local paralysis and disordered muscular action may be direct symptoms of hæmorrhage compressing or irritating recognized centres of muscular control; tetanic spasm is not infrequent, but clonic contraction are of rare occurrence, except as the result of an associated lesion.

The pupillary condition usually suffers some change, but none which is characteristic. Every possible combination of contraction, dilatation, and normal condition, with the single exception that contraction of one pupil never occurs without some change in its fellow, is associated with every variety and situation of hæmorrhage. Dilatation in some combination is more commonly observed than contraction, but not more frequently upon the side of the effusion than upon the opposite; and not more characteristically with one type of hæmorrhage than with another. In complicated hæmorrhages dilatation of both pupils is more common, and the effusion is more frequently bilateral than in the more nearly simple cases; and in unilateral dilatation is more likely to be on the corresponding side. A normal condition of the pupils is compatible with every variety of hæmorrhage wherever situated, whether simple or complicated.

Sensory disturbances, as delirium or irritability, are not symptoms of hæmorrhage, and when they occur are to be regarded as indicative of an accompanying lesion of the parenchyma.

*Subarachnoid Serous Transudation.* The serous transudation from the pial vessels which occasionally results from meningeal contusion can not be connected with symptomatic conditions.

*Arachnitis* is either acute or subacute in form, and is typically caused by a diffused meningeal contusion, though exceptionally propagated from a point of localized injury. It is sometimes an immediate result of the meningeal lesion, and it may be insidious in its inception and progress, but its beginning is usually late and is sharply defined. Its invasion is likely to be marked by a distinct and rather sudden elevation of temperature and an evident change in the general condition of the patient. The subsequent course of temperature is erratic, and the characteristic symptoms are those of cortical irritation. The pupils are oftener normal than otherwise, and changes in the characters of the pulse and respiration are slight. The form of the effusion is not necessarily reflected in the course and nature of the symptoms. The question of infection is uncertain.

General contusion is a constant complication of all other forms of intracranial injury, but rarely occurs as an isolated lesion of fatal severity. Its symptoms are irregular in their development, course, and termination, and indefinite in their mutual relation. This lack of conformity to any classical rule is due to the comprehensiveness of the lesion, its regional variations, and the fluctuations which occur from time to time

in the distribution of the movable fluids upon which its manifestations mainly depend. A loss of consciousness, at some time and in some degree, is more nearly constant than any other individual symptom, and the conditions of temperature are more uniform than any of the other phenomena which it occasions. The temperature is not likely to be sub-normal at the time of earliest observation, nor to exceed  $99^{\circ} +$ ; its subsequent course in cases of intensity is progressive, with few recessions, and ultimately reaches elevations of high degree.

Primary or early delirium, like primary unconsciousness, in both simple and complicated cases is to be ascribed solely to the influence of this lesion. The diagnosis must largely depend upon the recognition of the fact of intracranial injury, and upon the further possibility of excluding its other varieties, or, if they exist, of segregating the effects which they produce from a distinct remainder of symptoms.

Limited contusion is comparatively infrequent, and when it occurs in scattered areas through the centrum ovale, is not distinguishable from the general form of the same lesion; when it is cortical, it differs from laceration only in the extent of injury done to tissue; and symptoms, if they result, differ only in degree. It is, therefore, practically impossible to diagnosticate it from those lesions in their mitigated form.

Laceration is almost, if not quite, invariably complicated by a concomitant general contusion and by a resultant hæmorrhage. The primary loss of consciousness, and the delirium of some grade or character which often precedes or follows its restoration, are attributable to the attendant general contusion. In trivial cases there may be no secondary symptoms which indicate the fact of laceration. The primary unconsciousness may be replaced by a condition of lethargy or blunted perception, passing through somnolence into coma and death. The primary stage is most frequently succeeded by mental aberration or decadence, which may terminate in recovery, permanent dementia, or death. In exceptional instances consciousness may remain unimpaired, with extensive laceration of even fatal import. There is no necessary relation between the gravity or simplicity of the early psychic symptoms and the outcome of the case.

The temperature is higher than in any other form of intracranial injury, and, in cases destined to an early fatal termination, is characterized by a rapid and progressive increase, which sometimes continues for a certain time after death has occurred.

An irritability or abnormal sensitiveness to external impressions, often noticeable even after the supervention of final unconsciousness, and wanting in cases of hæmorrhage or contusion, is of frequent occurrence.

Convulsions, especially in implications of the frontal or temporo-sphenoidal lobes, are frequent in fatal cases, and so infrequent in the history of other lesions that they may be regarded as characteristic. The presumption that they are occasioned by laceration rather than by hæmorrhage is strengthened by a previous high temperature.

The loss of fecal and urinary control is common to all extensive lacerations without reference to the abrogation of consciousness or of muscular power. It rarely follows other forms of intracranial injury and is very nearly pathognomonic. The urinary and fecal discharges may be either unconscious or involuntary, or they may be the result of the patient's indifference to his surroundings. There are no demonstrated centres of control.

Paralyses are so much oftener the result of other lesions that they are of service only in determining the location of a laceration the existence of which has been already predicated upon more positive manifestations.

The pupillary changes have no greater diagnostic value than in hæmorrhages; the pupils are, in fact, normal in a much larger proportion of cases.

The characters of the pulse and respiration are habitually unchanged unless modified by the existence of complications. The contrast afforded by their substantially normal condition in an environment of pathic phenomena gives them the highest diagnostic value which they possess in this particular relation.

The bilateral variation in axillary temperatures and in the force and fullness of arterial pulsation, already noted as of unknown origin and referred to general diagnosis, is common to all forms of intracranial injury.

The manifestations of psychic disturbance are confined to cases in which the frontal lobes are implicated, but this implication is so constant as to make them practically symptoms of laceration in general. The other special symptoms which localize the seat of laceration has been already summarized.

The phenomena as indicated which directly point to laceration may be enumerated as certain peculiarities of temperature, psychic disturbances, loss of fecal and urinary control, and clonic convulsions.

Pyogenic parenchymatous inflammation is infrequent, and is of limited form, except when caused by the intrusion of a foreign body. Direct laceration and infection through the medium of compound fracture affords no question of diagnosis and is excluded from consideration.

The predisposing cause of traumatic central abscess is limited contusion; the exciting cause is supposed to be the admission of a pyogenic germ from some source external to the body. Though this supposition as to the source of infection may be correct, the further proposition that a route of entrance is always afforded by a superficial wound of the head is erroneous. Cases have occurred and are recorded in which no such wound existed.

The number of instances in which the histories of these limited pyogenic processes have been carefully observed or recorded is insufficient for the formulation of rules for diagnosis. The two cases which I have presented, and a third which I have noted, are in evidence of their uncertain symptomatology.

These conclusions are derived solely from the analysis of the cases which I have detailed, and are stated in as positive terms as the limited number of observations made will warrant. The series of cases presented, if insufficient to afford a basis for statistical inference, is yet so extended that the generalizations which it justifies are entitled to credence until controverted by results obtained from the study of a very much larger number of cases subjected to equally careful examination. It may be questioned whether deductions made, as in this instance, from the comparison of some hundreds of cases are likely to be materially changed by any subsequent multiplication of their number.

Symptoms are so diversified, their combinations so varied, and their continuance is sometimes so brief, that constantly careful observation and equally careful record are essential to thorough comprehension of intracranial injuries. If there are few symptoms which are intrinsically pathognomonic, there are many which by mutual relations of time and circumstance assume a pathognomonic character.

The possible multiplicity of lesions must be recognized, the relative as well as the absolute value of symptoms estimated, and if necessary some interval of time afforded for the development of the pathic condition; diagnosis becomes then neither more difficult nor more uncertain than in a majority of grave traumatic or idiopathic lesions. J. C.

**Triorial as an Hypnotic.**—Steiner (*Deutsche Med. Wochenschr.*, No. 13, 1895). The writer says, while, doubtless, the physician should preserve an attitude of reserve towards the many so called new remedies in the market, since business interests often overshadow their true value, it must be conceded that many a valuable remedy remains unused, because it has not been subjected to a thorough course of experi-

mentation. The necessity for a reliable and safe hypnotic is so urgent that every remedy recommended for this purpose should be given a trial, and if its efficacy be confirmed, the attention of the profession should be called to its merits.

With regard to trional, a drug related to sulfonal and which was discovered by Baumann and Kast in 1890, so many observations are at hand, confirmatory of its advantages, that there is nothing to prevent the general employment of this hypnotic.

The writer had previously made sulfonal the subject of experimentation, the results of which were published in the *Therapeutische Monatshefte*, October, 1889. In comparing trional with sulfonal, he unhesitatingly gives the preference to the former.

The hematuria which has been observed after long continued administration of both these drugs is of extremely rare occurrence, and usually preceded by constipation and oliguria. By attention to these conditions and by the employment of alkaline mineral waters, as recommended by Goldmann (*Therap. Monatsh.*, November, 1894), it can be prevented.

Aside from these rare sequelæ, trional is perfectly innocuous, both as regards the digestive, circulatory and respiratory functions, so that it is well adapted for pediatric practice. Dr. A. Claus (*Internat. Klin. Rundschau*, No. 45, 1894), has employed it in nervous affections of children attended with insomnia, especially in chorea, pavor nocturnus, convulsions, etc., in which it proved extremely serviceable. He administered it according to the age, in doses of 0.2 to 0.5 gm. in hot milk, confections or honey.

The writer employed trional in the case of a very decrepid lady, aged seventy-four, who suffered from emphysema and passive renal congestion, giving it in 1.0 gm. doses at night. The results were excellent, both as regards the insomnia and the diuresis which was markedly increased, and a condition of euphoria, such as had been long absent, ensued.

After Barth and Rumpel had made the first trials with this remedy at the Hamburg Hospital (*Deut. Medicin. Wochenschr.*, No. 32, 1890), their favorable results were confirmed by Schäffer (*Berlin. Klin. Wochenschr.*, 1892), and others. Stieglitz (*New York Medic. Monatschr.*, No. 12, 1893), employed trional in fifty-eight cases of various diseases attended with insomnia, while previous observations had been almost exclusively made in cases of mental diseases. According to Stieglitz the chief field of usefulness of the remedy is in simple agrypnia dependent upon cerebral neurasthenia or faulty habits of life. He found that in the agrypnia which is so frequently met with in hysterical women, especially during or shortly before the menstrual period, 1.0 gm. was usually sufficient to produce sleep of six to eight hours' duration. On the ground of his own observations and those previously published, Stieglitz concludes that trional is an hypnotic, especially suitable for general practice; and that as regards promptness and certainty of effect, ease of administration and freedom from disturbance it leaves much less to be desired than in the case of other hypnotics. Goldmann in a not inconsiderable number of cases of sleeplessness not produced by pains, especially neurasthenic insomnia, obtained sleep of six to eight hours' duration from the use of trional. Steiner's experience leads him to coincide in the conclusions of these authors. He emphasizes especially the rapidity of action of trional (ten to fifteen minutes) as compared with sulfonal which produces sleep as late as two hours after its administration, which is more often followed by dyspeptic symptoms, and more frequently proves inefficient.

1.0 gm. is usually sufficient; an initial dose of 1.5 gm. acts too powerfully in most instances, the patients complaining of marked lassitude on the following day and refusing further use of the remedy, while

the feeling of comfort produced by smaller doses constitutes a decided advantage of trional over other hypnotics. Trional is more soluble than sulfonal and is more readily absorbed. It should be administered in hot milk, tea or soups, and, in order to prevent a diminution of the alkalinity of the blood, one or two bottles of some alkaline mineral water should be taken daily.

J. C.

**Hereditary Ataxy.**—Fornario (*Annali di Neurologia*, 1891, Fasc. vi.)

A description of three cases of hereditary ataxy, with the following genealogical table :

Parents healthy.	{	Sister.	Five healthy sons.
		Sister.	Died unexpectedly in convulsions at the age of twenty-four.
	{	Brother.	Four sons healthy—one with infantile cerebral paralysis from birth.
		Father of the three patients.	Daughter of eighteen years, with Friedreich's disease.
Parents healthy.	{	{	Son of sixteen years with Friedreich's disease.
			Son, of twelve years, with Friedreich's disease.
	{	{	Three sons died in childhood from eclampsy.
			One died from an unknown disease.
{	{	Mother of the three patients.	Three sons of ten, eight and four years, healthy.
		Brother healthy.	One child of six years has crossed hemiplegia, paralysis of the right third nerve and of the left extremities with hemichorea.

CASE I.—May, 1891. Girl, 18 years old ; at the age of five years febrile disease, typhoid like in its symptoms ; when the disease was over disturbances of gait were noticed, which grew worse constantly ; inability to walk during past eight years ; since then speech also became slow, monotonous, drawling ; since from six to seven years' violent pain in the toes and legs ; since five years steadily increasing curvature of the vertebral column ; at present, headache and frequent attacks of vertigo.

Present condition.—Small short limbs, hands as small as the hands of a baby ; feet : long, straight ; kypho scoliosis of vertebral column with the concavity to the right side ; head deviates to the right ; short irregular oscillations of the head ; lateral nystagmus ; asymmetry of face, the left half smaller ; antero-posterior and lateral movements of the trunk impossible ; can hardly maintain herself in a sitting position ; impossibility to raise herself without aid to a sitting position ; with the upper extremities all movements possible, but show inco-ordination and are accompanied by irregular oscillations, increasing with the complexity of the movement performed ; motor power of hands rather well preserved.

Lower extremities.—Mobility limited to slight flexion on the hip ; no other movements possible ; knee-jerks and Achilles tendon reflexes absent ; plantar reflex and reflexes of mucous membranes present ; sensibility examined desultorily preserved in all forms ; slow, monotonous, scanning speech.

October, 1894.—Very marked inco-ordination of movements of the head, trunk, face, lips and tongue ; sensibility well preserved in the upper extremities, in the lower ones esthesiometric and thermal sensibility have disappeared, only sensibility to pain preserved ; cutaneous

and pupillary reflexes normal; specific senses normal; evacuation of bowels every four to eight days; no disturbances of bladder.

CASE II.—Boy, 16 years old, much exposed to humidity and excessive work; no alcoholic excesses; no onanism; no coitus; the disease began after a typhoid-like febrile attack; symptoms and course of disease similar to those of Case I. The chief points of difference are: No asymmetry of face; paralytic symptoms limited to the feet, but marked ataxy of the lower extremities; cannot stand without support. Paroxysms of pain in the nails, feet, calves and knees, extremely violent, more intense than in Case I., of a lancinating contusive character—at the beginning he could still perform the most complex movements with the hands and fingers (which was not possible in the first case), although accompanied by irregular rapid oscillations; knee-jerks first exaggerated, later diminished in one of them; *pied bot* of both feet.

CASE III.—Boy of 12 years; disease began in the same manner as in the previous cases at the age of six; nearly analogous to Case II, but *pied bot* of one foot only; paroxysms of pain unilateral; knee-jerks absent.

The three cases have in common:

The astasia, ataxy, tremor, nystagmus, the spastic rigidity, the deformity, especially of the feet, the long absence of disturbances in the function of the bladder and rectum; the familiar heredity; (the symptoms mentioned heretofore forming the complex of Friedreich's disease), the starting of the disease with febrile typhoid-like attacks, the slow development with symptoms of debility, the latter preceded by disturbances of coordination and followed by paroxysms of violent pain of a contusive lancinating character; the predominance of the motor disturbances in the lower extremities, the gradual but progressive development of sensory disturbances.

Special peculiarities of the first case were: The intensity and extent of the parietic symptoms; the arrest of development, if not atrophy, of muscles. The second case showed the peculiarity of exaggerated knee-jerks.

This exaggeration of the knee-jerks in one case would speak in favor of the view that this case was of cerebellar origin, while the absence of the knee-jerks in the other two cases would make a cerebellar anatomical substratum, probable for them. But otherwise there is such an analogy in the symptoms and course of the disease of all three cases that, considering the familiarity, "it seems unnatural" to assume that they had a different anatomical basis. The author finds that a clinical distinction between purely cerebellar or purely spinal hereditary ataxy on one side and cerebellar and spinal ataxy on the other side cannot be made. It seems to assume chiefly cerebellar origin for Friedreich's disease, as he calls attention to the resemblance between its symptoms and those observed after experimental cerebellar ablations. ONUF.

**A Case of Gerlier's Vertigo.**—Ackermann (*Centralblatt für innere Medizin*, March 2, 1895), describes a true case of this rare "neurosis." A laborer, 35 years old, was taken suddenly sick with severe pain in the neck, absolute ptosis of both eyelids, general weakness and loss of speech. After ten minutes the attack passed off. Similar spells were repeated at first monthly, but later more frequently. Between the attacks his health was good. There were no evidences of alcoholism, arterial sclerosis or other disturbances of the nervous system or of the digestive organs. There existed, however, a marked left amplyopia, apparently congenital. Under treatment by rest and faradization there was no improvement, but the attacks disappeared during the employment of warm baths and two grammes of iodide of potassium a day. This the author considers an argument against the hysterical or neurasthenical nature of Gerlier's vertigo.

FREEMAN.

**Dr. W. H. Baker** writes (*Boston Med. and Surg. Journal*, March 7, 1895) concerning the "Removal of the Uterine Appendages for Nervous Diseases." He outlines a somewhat hopeful spirit as to the effect of such removal, and gives two cases in which recovery followed, although a considerable time after the operation. He ends with the following summary:

"1. Diseases of the ovaries or fallopian tubes are sometimes an unrecognized cause of nervous disease.

"2. The adhesions resulting from attacks of localized pelvic peritonitis is a not infrequent source of nervous disease, either from the presence of pseudo-membranes in themselves, or from the interference which they offer to the functional activity of the organs involved.

"3. The extent or form of the pelvic disease is no indication of the character or degree of the resulting nervous manifestation.

"4. A most thorough pelvic examination should be made with the aid of an anæsthetic in every obscure case of nervous disease in a woman, occurring during the age of menstrual activity.

"5. Some forms of uterine disease may occasion an amount of nervous disturbance which may require the removal of healthy tubes and ovaries as the simplest and best mode of cure."

In the discussion Dr. Homans outlined his own cases, and expressed his belief in the curative efficacy of oöphorectomy as very slight. Dr. Edes and Dr. Davenport gave similar opinions.

Dr. Knapp said that he had never yet seen good results from it, and was "very doubtful about the cases of disease of the uterus, or ovaries, or any other organ of the body, giving rise to nervous disease, which does not at the same time give to certain symptoms, pointing to the organ itself." And again, "people are very glad to find some foreign cause rather than to admit the inherited or natural weakness of the brain." Dr. Prince expressed similar views, and particularly spoke against the too little lack of logical connection between remote slight causes and brain symptoms. He says farther concerning the symptoms themselves, "Certainly, the origin of many of these is not in the uterus, but in the mind itself."

PHELPS.

**Cerebral Anorexia.**—O. Seltmann (*Centralblatt für innere Medizin*, March 2, 1895). In anorexia it is important to decide whether we have to deal with true impairment of the sense of hunger or with a want of appetite, whether or not there is abhorrence for food ordinarily, and accompanied or not by vomiting or nausea. The dyspeptic anorexia, which appears as a symptom and a consequence of original visceral disease, comes on between the second and seventh year in pampered and spoiled children, especially among the better classes. Seldom is the anorexia of nervous origin in these cases, but it appears as a true neurosis without assignable visceral disease in children enumbered by neuropathic heredity. In a twelve-year-old boy reported by S. this condition appeared, and led to almost complete food abstinence, with the highest grade of inanition. Hospital care and gavage were without avail, but faradization of the head led to a surprisingly quick cure. The current was passed through the post-auricular region and also obliquely through the head daily, each sitting occupying, at the most, two minutes. S. believes that by these means the cortical circulation was so influenced that an improvement in the nutrition of the cerebral cortex was brought about.

FREEMAN

**Observations on Apoplexy.**—C. L. Dana, M. D. (*Med. Record*, February 24, 1895). The term apoplexy as used in this article includes not only cerebral hæmorrhage, but also embolism and thrombosis. From a study of 152 cases observed, D. states that one-third of the patients were females, and that the special apoplectic age was between forty and fifty years. Three-fourths of all the adults were between thirty and sixty. After the age of seventy it was relatively rare. Syphilis was a factor in

one-third of the cases. The special characteristics of the syphilitic attacks were that they occurred early in life, and more often relatively in women. Prodromata were frequent. The seizures were multiple in character, and the lesion usually a thrombus or softening. The fact that syphilis causes one-third of all cases of apoplexy has not heretofore been brought out, but most frequently the experience of neurologists will confirm it. In the cerebral hæmorrhages observed prodromata were rarely noted, and in many cases the patient even stated that he felt unusually well just before the attack. It was mostly in cases of thrombosis that prodromata were described. Hæmorrhages occurred in the morning oftenest, next, in the evening, and rarely during the day or in sleep. None of the fatal cases occurred during sleep, and D. doubts if arteries really rupture at this time. About one-fourth of those stricken died from the attack. Hæmorrhages were the most dangerous, thrombosis, especially specific, being the least so. While it was formerly taught that early apoplexies were embolic, the writer believes it more correct to call them syphilitic, and either thrombotic or hæmorrhagic. Excessive mental work does not lead to apoplexy among those who live temperately. The average duration of life for the survivors of one attack is over five years. Before the fourth year the chances of a second attack are always considerable, yet do not amount to fifty per cent., and are inconsiderable so far as hæmorrhages are concerned. Thromboses are much more apt to occur than hæmorrhages. The conditions of modern civilization tend to increase the number of cases of apoplexy, particularly those due to hæmorrhage. Under better sanitary conditions more people live to the apopleptic age, and the lessening of the number of infectious diseases increases the proportionate number of deaths from arterial diseases. There is also a larger urban population, with all that that implies in regard to the use of alcohol, the prevalence of syphilis, and the greater intemperance in eating and drinking. Apoplexy is regarded by Dana as a conservative agent, sometimes calling a halt to excessive activity and intemperate living and actually prolonging life.

FREEMAN.

**Unilateral Disturbance of Respiration in Cerebral Paralysis.**—Grawitz (*Zeitschr. f. Klin. Med.*, xxvi., p. 1).

An investigation of thirty cases of hemiplegia showed that in seven only was there no respiratory difficulty, and it is noteworthy that of these seven, six were females; and in all seven recovery was relatively rapid and complete. The respiratory disturbances accompanying hemiplegia are variable. In two cases typical Cheyne-Stokes respiration was present; in two others there was very marked diminution of respiratory movement and capacity on the paralyzed side. This diminution was noticeable, not alone on forced respiratory movements, but during tranquil breathing. This diminution seemed to consist, in some cases, of a retardation of the beginning of inspiration and a premature cessation of expiration. In one case of left side hemiplegia there existed a limitation of respiratory movement on the opposite side of the thorax; that is, the respiratory movement was much more energetic on the left side than the right. It is very remarkable that the paralysis of respiration disappears several weeks or months after the paralysis of the members. It would seem probable, therefore, that the cerebrum exercises the same action on unconscious respiration. The duration of these complications is too long to have them be attributable to an exertion on the respiratory centres by cerebral arrest.

J. C.



## Society Reports.

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### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, held at the New York Academy of Medicine,  
on Tuesday Evening, April 2, 1895.*

Dr. EDWARD D. FISHER, President, in the chair.

#### TUMOR OF THE BRAIN; SUCCESSFUL OPERATION.

Dr. C. L. DANA presented a young man, aged twenty years, who seven years ago received a blow on the left side of the head, in the neighborhood of the squamous suture. He was unconscious for a time, but soon recovered, and no immediate bad effects were noticed. Three years later he developed epileptic convulsions, which occurred about twice monthly and were general in character. About two years ago it was first noticed that the convulsions were preceded by a sensory aura, and commenced in the fingers of the left hand, then involving the arm, leg and face on the corresponding side, and finally becoming general. These attacks continued for over a year. He then came under Dr. Dana's observation, who found that he was suffering from a hemiplegia of the left side, involving especially the arm and to a lesser degree the leg and face. The paralysis was accompanied by various forms of anæsthesia, by intense headaches and by marked double optic neuritis. There was also severe pain in the left arm, and the patient's condition was very distressing. A diagnosis of tumor of the brain, affecting the left arm and finger centres was made, and Dr. Conway, on opening the skull in that locality, exposed a tumor lying just underneath the dura; it was flat and hard, and anteriorly it had infiltrated to some extent the cortical substance; posteriorly it was not attached to the cortex, but there were small whitish spots underneath the pia, denoting some infil

tration. It was found impossible to remove the growth in its entirety, and only about one-half was taken out. It proved to be a spindle-cell sarcoma. Since the operation, which was performed in May, 1894, (nearly a year ago), the improvement in the boy's condition has been very striking. For nearly six months after the operation he had no epileptic attacks at all; the pain and headache disappeared, as did also the optic neuritis. His left arm has regained considerable power, and he is able to move the fingers to some extent. During the past five months he has had about one epileptic attack each month, the movements being confined to the paralyzed side; they are not accompanied by loss of consciousness nor the severe pain he formerly had. Between the attacks he is perfectly well. Of course, the ultimate prognosis of the case is unfavorable, but the boy has had a year of comparative comfort and usefulness, and at present his condition is not growing worse. After the operation he was placed on arsenic, which was continued in moderate doses last summer. He has also taken some bromides. It appeared that the tumor developed from the dura.

In conclusion, Dr. Dana said that at a meeting of the Society last fall he referred to this case and gave the results of a careful study of the cutaneous disturbances. Immediately after the operation, the sensory disturbances seemed to be temporarily aggravated. At the present time there is only apparent a disturbance in the power of localization.

DR. SACHS—In regard to the patient shown by Dr. Dana, the speaker said he has seen several cases in which the excision of cortical substance for epilepsy, cyst or tumor resulted in sensory disturbances which were recovered from within a few days—much more rapidly than from the motor symptoms. The speaker also regarded Dr. Dana's case as one of the most successful among those reported after the removal of a cortical tumor.

Dr. W. H. THOMSON read a paper, entitled

## RELATIONS OF THE CEREBRAL CORTEX TO SENSATION (see page 333).

Dr. B. SACHS said he was a little surprised at the turn which Dr. Thomson's paper had taken. From the title of the paper he was led to expect that it would deal

with the question of whether or not there were true sensory areas in the brain cortex, and whether those areas overlapped or were entirely distinct from one another. Still, the paper brought up a number of interesting questions which are well worthy of discussion. It appeared to him that Dr. Thomson attached too much importance to the experiments that have been performed on the lower animals. The analogy between the lower animals and man should not be drawn too closely. There is a great difference between a dog and man: the former may be able to accomplish much without his cerebrum, while the latter can do very little if he is deprived of relatively large portion of it. In his paper, the author has invariably referred to sensory stimulation, or to a simple sensory impression as sensation, which is after all different from true sensation, into which the element of consciousness enters. Sensation, as applied to human beings, is not merely perception, but apperception.

Dr. Thomson seems to believe that epileptic seizures can occur without a cortex. This was disproven some years ago by Binswanger, who distinctly showed that while general tetanic contractions might occur in the body with lesions in the medulla or even below that level, orderly convulsive seizures never occur unless there is disease or irritation of the cortex.

Everyone is willing to admit that the brain of a child is a blank until sensory impressions are received, and that motor functions are the result of these. It is probable that we must sooner or later modify the conceptions we now entertain as regards cortical areas and their limitations. While the size of a certain area is, of course, of some importance, the immense number of cells present in each area must be taken into consideration. In the motor area for speech, for instance, sufficient number of cells are present to accommodate the wants of the poorest speaker and the most fluent orator, the latter simply taking possession of so many more cells.

The speaker expressed his conviction that the motor and sensory areas in the cortex overlap to a certain extent. He is willing to suppose that all the motor centres were primarily sensory, and that there is an actual representation of various kinds of sensation in the cortex. Dr. Thomson is probably correct in the assertion that the cortical centres are not as strictly limited as we suppose, but in the main we should base our deductions upon the experimental physiology that has been

conducted through the surgeon's work in this field, and be guided by those results.

Dr. C. L. DANA said he would confine himself to a statement of what he considered to be the facts in regard to the localization of sensations. He looked upon a sensation as the earliest and simplest of the conscious states. If the end of the finger is touched by some object, we get a sensation of contact. If a white light is thrown into the eyes, we get a simple sensation of light. When the contact is felt to be rough and cold, and association and memory are brought into play then we get concepts. If we attempt to make the term sensation mean anything more than that indicated we are thrown at once into great confusion. The speaker believed that simple sensations of touch, smell, sight, hearing, etc., were well differentiated and localized in the brain cortex, nor did he believe there were any truly conscious states in man except in connection with the activity of the cerebral cortex. If we shave off the motor cortex on both sides of the human brain, we produce cutaneous anæsthesia, and then no kind of contact will arouse any sensation of a cutaneous character. We might get reflex acts, but they would not indicate that any sensation was present. The same doctrine probably holds true regarding the special senses of sight, smell, taste and hearing.

In connection with this subject the following case is of interest: A child was born at Bellevue Hospital and lived for five days, during which time it cried when it was hungry or when it was pinched or roughly handled; it winked its eyes and showed certain manifestations of apparent intelligence. At the autopsy it was found that the cerebral hemispheres were absent; there were merely small fragments of the corpora striata and imperfect optic thalami. In that instance there were probably no genuine sensory impulses; the impulses were simply excito-reflex in character. Sensations, carefully defined and understood, and rigidly separated, will probably be found to be conscious units which are localized in different parts of the cortical area. As regards the point brought out by Dr. Thomson that motor cells never explode or discharge themselves without being first "touched off" by an afferent impulse, the speaker said he was quite ready to believe that that was possible, or even always the case.

Dr. W. R. PRITCHARD said that he expected from the title of Dr. Thomson's paper that it would deal with the

question of cortical representation of common sensations; he had no idea it would take the psycho-physiological turn it did. Personally, he believes in the doctrine of cortical representation for common sensations, and furthermore, that the cortical regions in which these sensations are represented, correspond very closely with what is known as the motor area. He had hoped that in the discussion some reference would be made to other sensory functions which are not included under the term of common sensations, but are more particularly related to the viscera, the heart, lungs, stomach, etc. These sensations have been variously styled automatic or reflex in character, but he is of the opinion that they are distinct, conscious impressions, just as much so as are the sensations of touch, temperature, etc. He has collected certain facts touching on this subject which he expects shortly to bring out in a paper, which facts are suggestively confirmatory of a theory of cortical or at least cerebral, representation and definite localization of these sub-divisions of sensation.

Dr. JOSEPH COLLINS said that to his mind, the facts brought forward by Goltz and others who have experimented on the lower animal were not inimical to the contentions held by those who believe in the localization theory. The movements of those animals, such as balancing, coordination, maintenance of equilibrium, and even the remarkable efforts of prehension and apparently purposive movements, can be explained on purely physiological grounds, and do not have to be taken higher up into the realm of the sensorium than the medulla or perhaps one plane higher; at least caudad to the internal capsule. He inquired of Dr. Thomson how he reconciled his conception of the primary and secondary neuron with his position in regard to the non-necessity of anything higher than the spinal cord for the possession or maintenance of the sensory and motor function, sensations, etc., as referred to in his paper. In that case, what functions does the primary neuron perform? Dr. Thomson attributes to the secondary neuron all the important phenomena which are ordinarily attributed to the cortical structure and its ramifications.

In regard to the concluding portion of the paper, Dr. Collins said he did not think that in epilepsy there is a surcharge of energy in the cortical cells, which explodes, but on the contrary, on account of their pathological condition the cells are unable to retain securely an

amount of energy which they are able to hold in their physiological condition. That is, they lose their tenacity and an explosion results.

Dr. E. D. FISHER.—The great difficulty in discussing this subject is to fully understand the difference between direct afferent impressions and true sensation. When we speak of sensation ordinarily, we mean the perception or appreciation of a certain thing by the person or animal. I believe that animals have true sensations; the dog is not only able to appreciate pain, but he is also able to appreciate comfort and discomfort. The sensation of pain must be received somewhere in the cortex; just how definite its localization is, it is difficult to say. It seems to be necessary that the sensation come from the external portion of the body, for, if we irritate the cortex itself, it does not give rise to pain. In its efferent or motor aspect, however, the cortex responds, whether the irritation comes from without or within. A scar on the finger may give rise to an epileptic seizure, beginning in that finger; a similar attack may be induced by irritating the brain centre governing that region of the body.

Dr. THOMSON.—I apologize for having misled the gentlemen who took part in the discussion by the title of my paper; my idea in bringing up the subject as I did was to simplify it. If we begin at the cortex, we are confronted with the most complicated end of the problem; it, therefore, occurred to me to begin from below, going up towards the cortex, and then determine whether such a thing as sensation could properly be said to be found before we reached the cortex.

I agree with Dr. Sachs that we must be careful about interpreting facts observed in the lower animals, and applying the deductions to man; but when it is a mere question of intelligence, I see no reason why an analogy should not be drawn between dogs and man. Many dogs seem to possess more intelligence than monkeys, and the latter are supposed to be very closely related to man. The results of Goltz's experiments are certainly very remarkable.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, March 25, 1895.*

Dr. JAMES HENDRIE LLOYD, President, in the chair.

Dr. THEODORE DILLER, of Pittsburg, presented a paper on

### A CASE OF JACKSONIAN SENSORY EPILEPSY.

#### DISCUSSION.

Dr. CHARLES K. MILLS. Dr. Diller, in sending this patient, asked particularly for suggestions concerning treatment. In the first place with reference to the nature of the case. It seems probable, as Dr. Diller suggests in the paper, that this atrophic condition has been going on for years and that it is of the nature of a facial hemiatrophy. It is likely that his injury has had had something to do with his recent manifestations—with this Jacksonian sensory epilepsy, if we choose to call it that—and with the motor attack which he has had at least once. It is difficult to get a clear idea of the mechanism of these attacks. The suggestion which Dr. Diller makes is a reasonable one, particularly in the light of Mendel's report, and we have, perhaps, had one or two similar reports. If he has atrophy of the terminal nucleus of the fifth, in the region of the locus ceruleus, and if there has been an injury to the fifth nerve fibres in the scalp, it may be that we have a hint as to the mechanism of these attacks. Given an atrophic region, which is always unstable, and a peripheral irritation of the fibres of the first conveyed back to its nucleus which is close to the lemniscus in the pons, it may be that this is the starting point of the trouble, and that it is thus that the impulses are projected to the sensory cortex, and completing the cortical arc to the motor attacks. You have thus a theoretical explanation of the case. Of course, it is speculation, but it is difficult to explain this case. Certain other phenomena are very striking, as the fact that after these well-described attacks, he has headaches persisting for minutes or hours. This in connection with the localizing symptoms probably shows some wide-spread cortical

involvement. If this be a correct theory, it might be worth while to try the effects of operation, at least exploratory operation, in the neighborhood of where the scalp was injured. The question of the possible hysterical nature of these attacks might be considered, but I would rather hear what the President has to say on that point.

The PRESIDENT.—It seems to me that we have here a typical hemifacial atrophy of rather short duration. According to the history of the case, this hemifacial atrophy has probably been coming on for ten years, although it has been marked only for the last few years, following the kick by the mule. We know that in the main these hemifacial atrophies do not have a history of trauma, and I think that we can not ascribe this case to trauma, for it undoubtedly began before the traumatic impression was made.

From my knowledge of hemifacial atrophy, I do not see how we can connect it with the other nerve symptoms which the patient has. These nerve symptoms seem to be of a very bizarre type and, so to say, unsatisfactory character. We can not exactly tell what they are from the patient's description. On one occasion, he had a sort of pseudo-epileptic attack, but it does not seem to have been repeated or to have been at all characteristic. These subjective sensory disturbances, I think, cannot be connected with the hemifacial atrophy. They may, however, have had an origin in the trauma.

I think that in this case there should be a careful testing of the tactile and thermal sensation after these nerve-storms. A great deal depends upon such testing carefully done at the time of and after these paroxysms. There should also be a careful study made of the color fields. I think that the diagnosis of traumatic hysteria has not been eliminated. I do not see any indication for operation. If done, I think that such an operation would be of a mere experimental kind. I do not see how any operation on the fifth nerve could stop the progress of the hemifacial atrophy, and I do not see where the localization exists to serve for an indication for operation to control these sensory symptoms.

Dr. AUGUSTUS A. ESHNER presented

#### A CASE OF PSEUDO-MUSCULAR HYPERTROPHY.

Paper of Dr. Eshner, read by the Secretary.



Dr. DAVID RIESMAN read a paper on  
MERYCISMUS OR RUMINATION, AND PRE-  
SENTED A CASE (see page 359).

## DISCUSSION.

Dr. WHARTON SINKLER.—There is no question that rumination is much more common than is generally believed, and if we look into the matter more we shall find a greater number of instances of the disorder. I have met with a few cases, but none so typical as those detailed by Dr. Riesman. The cases which I have seen have been those of regurgitation of food without effort after meals. All the cases which came under my notice have occurred in thoroughly neuræsthenic women or men, and I cannot help coming to the conclusion that the affection is a neurosis and connected with a condition of profound neuræsthenia. I have no doubt that the influence of the will largely affect the act of rumination. One young lady who was under my care had been in the habit for years, whenever she felt inclined, of emptying the stomach, but she never had any inclination to ruminate in the manner of cattle.

Trephining has been suggested and even practiced for the relief of this condition.

The PRESIDENT.—When on duty at the Institute for Feeble-minded Children at Elwyn, I learned there of a number of cases that had the habit of merycism. In the case of one of the children the habit was peculiarly disgusting. He not only regurgitated his food, but disgorged, and then took it in his hand and replaced it in his mouth and re-chewed it. That, I think, is not usual in cases of merycism. Cases seen in idiots I think are rather of this character.

I have no doubt whatever that these cases are allied to the neurotic or neuræsthenic temperament. Dr. Sachs, in discussing this matter, has referred to a German physician, a personal friend, who had reported his own case. He overcame it by a persistent effort of the will.

If trephining ever cured a case of merycism it was merely by mental impression, or suggestion: but I do not think that it is necessary to cut a hole in a man's skull in order to put a suggestion into his mind.

Dr. CHARLES K. MILLS presented a note on

A CASE OF UNIFORM ARREST OF DEVELOPMENT OF ONE LOWER EXTREMITY WITHOUT ANY ACCOMPANYING ABNORMAL CONDITIONS.

I have seen several of these cases, and they sometimes in infancy are supposed to be infantile cerebral, or spinal palsy. They need to be differentiated from such cases. This patient is a little girl. The left leg is uniformly smaller than the right, yet perfect in every other way.

It is a matter of some interest as to the method in which these cases originate. In accordance with Hughlings Jackson's idea that large ganglion cells are related to large muscles and small cells to small muscles, it would seem that in such cases the cortical and cornual cells having started to develop at a certain pace, as regards size,—a pace different for the two sides of the neuraxis—have continued to develop relatively at the same rate at which they began. The consequence is, limbs which are normal on both sides but of different bulk.

DISCUSSION.

Dr. J. MADISON TAYLOR.—This leads me to refer to the case of a boy with depressed fracture of the skull exhibited by me in January, in whom an operation over the motor centres of the right side was followed by symmetrical failure to develop size in the left hand and arm, but perfect power was retained.

Adjourned.

*Stated Meeting, April 22, 1895.*

Dr. JAMES HENDRIE LLOYD, President, in the chair.

Dr. J. TORRANCE RUGH and Dr. CHARLES K. MILLS read a paper on

A CASE OF PROGRESSIVE MUSCULAR ATROPHY, SHOWING ESPECIALLY ATROPHIC PARALYSIS OF THE RHOMBOIDEI AND SUPINATI MUSCLES, THE TONGUE AND THE FACE (see page 354).

Remarks by Dr. MILLS.—The case is of interest from

several points of view. In the first place, it shows the same history that many of these cases do,—a history of which I do not understand the meaning, namely, that they not infrequently begin with attacks of pain in the region of the muscles which are affected. Then there will be at intervals similar attacks of pain with somewhat rapid increase of the muscular degeneration. Occasionally, cases similar to these are due to gliosis. In the present instance we could find no sensory changes. I believe that this case will steadily progress as have others that I have seen.

Dr. ELIZABETH G. BUNDY exhibited

A BRAIN, SHOWING AN OLD CYST, INVOLVING  
CHIEFLY THE THIRD FRONTAL AND THE  
LOWER EXTREMITY OF THE CENTRAL  
CONVOLUTIONS.

Dr. JOHN K. MITCHELL read a paper on

HUNTINGTON'S CHOREA.

Freeman Tucker, white, age 28 years, reported to Dr. Mitchell's clinic at the Infirmary for Nervous Diseases, March 29, 1895.

He gives the following history:

*Family History.*—His father suffered from rheumatism, and his mother died of results of Huntington's chorea, having been affected for over twenty years. There is no other history of neuroses or psychoses in the family.

*Previous History.*—Patient has always been healthy. In childhood he only had whooping cough and measles, and these in mild forms. He denies venereal infection. He states that five years ago he had a general muscular aching through the body. No fever accompanied the condition. Patient has not been able to work for two years on account of chorea.

*The present trouble* dates back eight years. No cause is assigned, excepting heredity.

His right arm began to move first in a choreic manner. The head gradually became affected, then the neck, and finally his speech became "thick" and the tongue uncontrollable. He was never paralyzed, but is often weak in the legs, and easily fatigued.

The trouble has had no decided exacerbations, but has gradually progressed to violent choreic movements, quite general, but worse from the pelvis up. These only cease during sleep, which is usually undisturbed.

His digestion is good and appetite fair. Heart sounds normal; pulse eighty when lying abed. The lungs present no physical signs or symptoms of disease.

Dr. Thomson reports: "Pupils react normally; disc and fundus normal; muscle balance is normal; low hypermetropia."

*Reflexes.*—Knee-jerks large and easily reinforcible; no clonus. Dynamometer, R. 75; L. 80.

Slight "crossed knee-jerk" exists. (Taylor & Hinsdale.) Cremasteric ÷: plantar, epigastric, abdominal and gluteal reflexes all give quick response. Muscles jerks all over the body are increased, especially those of trunk and upper extremities.

Sensation is everywhere acute; tongue clean and protruded slightly to the right side. On opening mouth widely it assumes an oval shape, due to spasmodic contraction of left levator-alae nasi muscle. Volition quiets movements a few seconds. Rest alone (except sleep) does not quiet much, and he tosses about violently with his arms in bed. He is not melancholic (nor was his mother), quite different from most reported cases.

Blood count April 5th, showed hæmoglobin 95%; red blood corpuscles 5,200,000; whites normal. The movements to-day are of head, face, lips, tongue, arms, hands, muscles of back and abdomen. Great difficulty of speech and of writing. Patient has a peculiar "grunting." Gait waddling and irregularly choreic.

#### DISCUSSION.

Dr. WHARTON SINKLER.—This is evidently a typical case of hereditary chorea, but in this instance, the facial muscles are much more affected than is usually the case. The gait also is not characteristic of Huntington's chorea. In this affection the patient will walk two or three steps quite naturally; then there is a choreaic movement which arrests the walk for an instant; another step forward is then made and he hops up to it like a dancing step and then again goes on naturally.

This form of chorea was not recognized by the profession at large until Huntington described it in 1872, although it had originally been pointed out by Waters

in 1842. In the first forty years only eight or ten cases were recorded, but in the last ten years a large number of cases have been reported. There are now in the Philadelphia Hospital five or six typical cases of chronic adult chorea, most of which are of the hereditary type.

The PRESIDENT.—I would ask Dr. Sinkler what connection he thinks that there may be between this class of cases and those which occur in pregnancy. The latter cases sometimes run a chronic course and present exactly this appearance. They have no hereditary history. They begin as an acute essential chorea and sometimes pass into this chronic form, persisting after confinement. I remember one case which ran into insanity. Another case has been choreaic for five years. I would also ask Dr. Sinkler if he has observed mental derangement as a common sequela of Huntington's chorea.

Dr. WHARTON SINKLER.—I do not think that we can distinguish clinically between cases of chronic chorea of pregnancy and hereditary chorea. The symptoms are almost identical. I believe that pathologically we shall find the same changes, evidently some cortical changes. These forms of chorea are due to organic change, differing in that respect from Sydenham's chorea, and the cortex is undoubtedly the seat of disease as has been shown by several post-mortem examinations.

Speaking of the chronic chorea of pregnancy I recall an interesting case in a young woman in whom the disease continued for several years. She became pregnant and the movements were greatly exaggerated, but after delivery the movements ceased and she has been well ever since.

In answer to Dr. Lloyd's question, I would say that nearly all cases of Huntington's chorea terminate in dementia.

Dr. WHARTON SINKLER read a paper on

### NERVE SUTURE.

He presented a patient in whom an operation for suture of the musculo spiral nerve was successfully performed three months after complete section of the nerve. The patient was a man, twenty-six years of age, married, intemperate as to the use of stimulants. In the early part of February, 1864, he was stabbed with a small blade of a penknife just above the left elbow. The

musculo-spinal nerve was severed, and immediately following the injury there was complete loss of motion in the extensors of the wrist and fingers. There was a sensation of formication in the thumb and forefinger for a day or two after the injury, but this soon subsided, and no loss of sensation was noted. The patient was first seen by Dr. Sinkler about ten weeks after the division of the nerve. At that time there was complete wrist-drop of the left hand, and inability to extend any of the fingers. The hand was much swollen, and on the dorsum of the hand was the hump which is characteristic of extensor paralysis. The distal phalanges of the fingers could be extended by the interossei muscles. There was no loss of power in the flexors, but owing to paralysis of the antagonist muscles there was inability to flex the fingers with any strength, so that the patient could not move the dynamometer one degree. There was no loss of tactile sensation, with the exception of a small area on the posterior and inner aspect of the thumb extending from the carpo-metacarpal articulation of the last phalangeal joint. In this region sensation was impaired, but not entirely lost. The muscles of the forearm and the first interosseous were wasted, and gave no response to the strongest faradic current that could be borne; to the galvanic current the reaction of degeneration was present. The hand muscles responded normally to the electricity.

On May 15th, three months after the injury, Dr. W. W. Keen cut down upon the musculo-spiral nerve and united the severed ends. The nerve was found to be markedly bulbous at the point at which it had been severed, the bulb apparently consisting of connective tissue. The bulbous portion was excised, and the two extremities stretched by the thumb and finger, and elongated sufficiently to bring them into opposition. The two ends were then sutured with two fine silk threads.

The excised portion of the nerve was examined by Dr. D. Braden Kyle, who reported that he found the severed portion of the nerve about three-fourths of an inch in its greatest diameter and one-half of an inch in its smallest diameter, the greatest diameter being in line with the trunk of the nerve. Microscopically the bulbous portion of the tissue appeared more fibrous than the portion of the trunk attached. On section microscopically it presented the same appearance. Under the micro-

scope, above and below the bulbous portion, the tissue presented much the normal appearance, except its reaction to stain, the cells seeming to be swollen or undergoing some infiltrating process, the nerve-sheath being thickened. In the bulbous portion partial longitudinal section of nerve filaments could be demonstrated, but the main part of the bulbous portion was composed of fibrous connective tissue. Distinct nerve fibres were present in the bulbous portion, but these fibres were widely disseminated, and were not arranged in bundles.

On May 28th, two weeks after the operation, an examination of the patient was made. There was no change in the paralyzed muscles, and the electrical reactions were the same as they had been in the previous examination, which was made on April 15th. The use of galvanism was begun, and steadily persisted in, with the addition of massage later. There was no evidence of return of power until the latter part of October, five months after the operation. At this time the patient began to have slight power to extend the wrist. There was gradual restoration of power, and on December 26th, that is, seven months after the operation, the patient was able to extend the wrist fully and strongly. There was full power of extension of all of the fingers. With the dynamometer the grip of the right hand was 140, with the left 65. At this time the extensor muscles of the left forearm still failed to respond to the faradic current, and with the galvanic current the anodal closing contraction was about equal to the kathodal closing contraction. A strong current was necessary, however, to cause the muscular contractions.

On April 9, 1895, an examination shows an increase in the muscular power, and an interesting change in the electrical reactions. The muscles still fail to respond to the faradic current, but there is also loss of reaction to the galvanic current with the strongest current available. A very strong galvanic current causes slight tetanic contractions of the extensor carpi radialis.

Dr. Sinkler remarked that the case was interesting for a number of reasons. First, because it showed the advantage of nerve suture after an interval of three months, and the importance of thorough and long-continued electrical treatment to the nerve and paralyzed muscles, even when but little encouragement had been obtained after several months treatment. The electrical reactions are also important. When the patient was first

seen, the reaction of degeneration and complete absence of response to the faradic current showed that there was entire division of the nerve, and the fact that restoration of voluntary power before the return of the normal electrical reactions is interesting, because it bears out the experience of those observers who have given detailed accounts of cases after nerve suture. A point of especial interest is the effort which had been made in regeneration of the nerve. The fact of nerve filaments being present in the excised bulbous portion showed that the process of regeneration had begun, and it seems probable that in time that this process would have become more fully developed, and possibly have restored the function of the nerve.

#### DISCUSSION.

Dr. JOHN K. MITCHELL.—In the last two years I have seen fifty-five cases of section of nerves, some sutured and some not, and I have examined the notes of over two hundred and fifty cases of sectioned nerves with more or less complete recovery. I deny the possibility of primary union. I believe the cases that have been reported as instances of primary union have been cases where sensation alone has been studied and not motion. The return of sensation has depended upon anastomosis and not upon reunion of the out-nerve.

The most interesting practical point is the recognition of the great success of secondary suture in this case. I believe that secondary suture, even at a much later period should be tried and that it would many times be successful. I think, too, that in favorable cases motion will be found to return as soon after secondary suture as after primary suture. The degeneration which follows every section necessarily occupies many weeks, and these weeks might as well pass without the nerve being united.

Dr. WHARTON SINKLER.—The time which intervened in this case between the injury and the suturing was only three months, which is only a moderate length of time. Cases have been reported where longer intervals have elapsed. In one case the sciatic nerve was united nine months after its division by a scythe and there was complete restoration of function.

Adjourned.



THE  
**Journal**  
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**Nervous and Mental Disease.**  
  
**Original Articles.**

THE DIFFERENTIAL DIAGNOSIS BETWEEN  
LOCALIZED NEURITIS, RHEUMATISM AND  
SOME OTHER CONDITIONS THAT GIVE SIM-  
ILAR SYMPTOMS.<sup>1</sup>

BY LOUIS FAUGÈRES BISHOP, A.M., M.D.

New York.

THE object of the study which led to this paper, was a more definite classification of the means of differentiating pain in the extremities. Only as a matter of convenience, and to bring the discussion more in line with the work of this section is neuritis made the central idea. Excluding pains in the extremities due to obvious local disease, there are a large number of cases in which a conclusion as to a cause is only to be reached by study and analysis. In this we must consider neuritis; we must discuss and determine the scope of rheumatism and gout, and we must attempt to give neuralgia a definite place. Of the causes in the central nervous system locomotor ataxia may serve as a type, though neurasthenia must be considered. A final class of pains will include those due to blood conditions, such as anemia, the products of disordered digestion, malaria and infectious diseases.

Pain.—Of the nature of pain itself we are quite in the dark. Its perception is a function of the nervous sys-

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<sup>1</sup>A paper read before the Section of Neurology of the New York Academy of Medicine, May 24, 1895.

tem, but how to draw the line between ordinary sensation and pain we are unable to say. As an accompaniment of inflammation it is most common, but we have pains without inflammation as neuralgia and we have inflammations without pain, as often occurs in inflammation of the mucous membrane. It is this indefiniteness of our knowledge of the occasion of pain that makes it so hard to classify these different local pains, especially those that are not dependent upon an ascertainable inflammatory state.

This question of the meaning of pain is one that so far has not given itself to very scientific investigation. In fever we have learned to measure height, duration, and even distribution to various parts of the body. In the very nature of things, pain being a subjective sensation, we have not been able to compare intensity with any known standard, nor have we been able to define quality with any degree of accuracy. In spite of the attempts so far made we are reduced to a few comparisons. We roughly estimate pain as slight, moderate, severe, intense, and so on into the superlatives, but each adjective is subject to the personal equation, habits of speech, and the susceptibility to pain of the patient. Pain is mobile, shifting, or darting, or it is fixed in one spot. As to quality, we use such adjectives as dull, boring, lacerating, pricking or crushing, our favorite adjectives being those which describe some form of injury. As to duration of pain we are better off. We can define pains as constant or intermittent. We might record accurately its duration, the time of day during which it is present, and its relation to attending circumstances, such as weather, certain forms of exercise, ingestion of food, etc.

It is conceivable that by and by we may have charts upon which all these things can be recorded and reduced to mathematical curves. The intensity and character of pain it will always be difficult to classify, but may we not discover means whereby we can produce "physiological" pains of a certain character and a certain degree of intensity, so that the patient can compare the quality and severity, and give us an idea as to its comparison with the pathological pain? For instance, a certain degree of pressure on the supraorbital nerve would produce a unit of pain which we could reproduce in ourselves if necessary, and in that way obtain a very clear idea of just what our patient was suffering. Severity

can be judged with a fair degree of accuracy by the effect it has upon the routine life of the individual; whether it is severe enough to prevent sleep or the ordinary occupations of life, or to cause vomiting.

As a foundation for the study of pain in the extremities it is necessary to have more than an indefinite idea of the anatomy and physiology of the structures of which they are composed. Physiology is nearly synonymous with function. Disease may or may not cause pain; it may or may not interfere with function; but it will almost always do one or the other.

*Neuritis*.—Not a great many years ago diseases of the peripheral nerves were little thought of. Since they have been recognized they have attracted a good deal of attention. So much so that just as in old times physicians thought of most pains as rheumatism or gout, so the temptation is now for the neurologist to explain many painful conditions by pre-supposing the existence of neuritis.

Neuritis is a common cause of a unilateral pain in the arm or in the leg, generally the result of traumatism or constitutional disorder, as alcoholism, syphilis, or of the gouty or rheumatic diathesis. It is characterized by its fixedness, its distribution to the territory of particular nerves, by the accompanying tenderness of the nerve trunk, absence of fever, and by the pain being worse at night. In addition to this other functions of the nerve may be interfered with. In diagnosis a good deal must depend upon the distribution of the symptoms as to whether or not they correspond to the anatomical distribution of a particular nerve, and upon the application of certain tests. Pressure upon the nerve trunk at the point where it is inflamed gives pain at the point of pressure, and increases the pain which previously existed in the area of distribution of the nerve. Numbness and tingling are frequently present over the areas of distribution of the terminal fibres of the nerve. Hyperaesthesia may be very marked and paralysis may be present when the nerve effected is a nerve of motion as well as of sensation. Muscular tenderness, as a symptom of neuritis, is one of a good deal of importance. It is more apt to be found in the beginning of acute cases, but may persist throughout the course of a chronic case. The low grade local neuritis, which we have in mind, is essentially a chronic condition. Its origin may be often obscure, or it may be traced from some slight injury.

Neuritis is not an uncommon condition as complicating the advanced wasting stages of tuberculosis and diabetes. In a good many cases of tuberculosis it goes on to toe drop and wasting of the legs. But in wasting diseases there is also a more obscure form of neuritis, which is sometimes very troublesome, affecting a single nerve or branch and being an occasion of much suffering. Examples of localized neuritis due to the pressure of inflammatory products, or new growths, are very common in surgery. Perhaps the most trying of these is the extremely painful condition which sometimes follows in a hand and arm that has been the seat of a severe cellulitis.

The onset of certain forms of neuritis is extremely hard to understand. Why it is that a person exposed to a slight draught develops a severe attack of facial palsy, the severity of the disease being entirely out of proportion to the apparent cause, is mysterious. May it not be that there is a deeper cause, such as rheumatic poison, circulating in the blood, and that the local chill has been the occasion of its disposition at a particular point, or else we may suppose that for some reason that particular nerve has been weakened, and rendered liable to inflammation. The pathological anatomy is well-known to differ in different forms of neuritis. In the more chronic and localized forms it is the connective tissue of the nerve that is mostly involved.

*Rheumatism.*—By the object of this paper (classification of indefinite pains) we are excluded from the pleasure of drawing pictures of well-marked cases. We hope to extend the scope of rheumatism to the fullest justifiable limits rather than restrict it. Rheumatism in its chronic and sub-acute forms does not confine itself to the fibrous structures of joints, but is prone to involve the intermuscular septia, the aponeuroses and the muscles. It has been the fashion to call muscular rheumatism myositis, but for our present purpose we include this in rheumatism. Rheumatic pain is very liable to be worse at times. It is affected by temperature and weather. The joints are very liable to show inflammatory signs, and are apt to be affected in succession. When so affected there is stiffness, especially after resting. The pain of muscular rheumatism at the outset is apt to be severe. It is affected by movements of the muscles. The pain is elicited by stretching the muscles by passive motion. Its distribution corresponds to that of the muscles, and

pressure on the affected part gives relief. It is a transient affection as a rule, though frequently enough is it more chronic.

All cases of subacute rheumatism carefully observed show slight rises of temperature. Indefinite stinging pains in middle-aged people suggest gout. We may presuppose their cause as due to the sudden precipitation of minute particles of urate of soda in the tissues, which after remaining for a short time may be re-dissolved. Our diagnosis must depend upon concomitant signs of the gouty diathesis.

*Neuralgia.*—Neuralgia is uncommon in the extremities. The pain intermits and when absent there is no tenderness of the nerve trunk.

*Disease of central nervous system.*—In posterior sclerosis pain may precede other symptoms by many years. The pains are severe and momentary, usually in paroxysms, but may be continuous. They sometimes distinctly follow the course of nerves. They are commonly felt severely in the neighborhood of joints. There is some relief by pressure.

*Neurasthenia.*—The pains of neurasthenia may occasionally present difficulties. The tendency is to attribute too many of these pains to this cause. Occasionally very bad blunders are made, and it is only upon the supervention of paralysis, or the discovery of some other serious condition that the mistake is discovered. One experience is enough to make one very cautious. Very early in my medical studies I had the opportunity of observing the case of a man who complained chiefly of a severe pain in the chest. From many concomitant symptoms and circumstances it was thought that the man was a malingerer. There were absolutely no abnormal physical signs. The man was treated with very little consideration, and finally passed from observation. A few months later I encountered the same patient with a pulsating tumor of the thorax due to aneurism which finally ruptured into the pleural cavity. At the autopsy the bodies of several of the vertebrae were found eroded. This case, seen as a student, made a very profound impression, and from that time on lead to a different interest in the study of the meaning of indefinite pains.

*Pains due to blood condition.*—The general aching of the extremities due to blood conditions is usually easily diagnosed by the exclusion of local signs. Those due to disordered digestion often have a definite relation to

the ingestion of food. The pains at the beginning of infectious diseases are accompanied by fever. In chronic metallic poisoning producing pains, there are usually other symptoms present.

*Differentiation.*—*Neuritis and articular rheumatism.* The differential diagnosis between neuritis of a severe well-marked type and well-marked cases of rheumatism or gout ought to be easy, but the class of cases of which so many are seen in dispensaries and private practice with localized pain in a limb, rather persistent in character, require a careful study of the attending conditions to make up our minds whether the nerve is primarily affected by a neuritis, or whether the nerve is simply giving expression to the pain caused by the disease of the tissue to which it is supplied.

However, mistakes of observation between neuritis and rheumatism are not confined to these mild cases. They may occur in the severest type of cases while first under observation. I recollect a case, the history of which I mean to look up more definitely, of a patient who was discharged after a number of weeks from a prominent hospital as a case of chronic rheumatism. This patient was admitted under protest to another similar hospital where attention was especially attracted to the case by the appearance of a bed sore. The man presented the typical appearance of a case of chronic rheumatism with the disability that often follows in old cases. However, a more careful analysis of the case led to the belief that it was a case of multiple neuritis, of gradual onset and obscure origin. The diagnosis was subsequently confirmed, and a complete recovery took place after a considerable length of time. This case is quoted because the mention of the subject of this paper has been the occasion for the remark that any one can easily tell the difference between neuritis and rheumatism.

In his work on "Diagnosis," Musser says, "I have had the pains of neuritis attributed to rheumatism of the phalanges, tarsus, and ankle, until paralysis of extension took place; and neuritis of the circumflex mistaken for shoulder-joint disease. Multiple neuritis is attended by pains that may be located in the joints by the patient, but whether local or general neuritis, the joints are never swollen, tender or painful on movement by the hand."

*Neuritis and aponeurotic and muscular rheumatism.*—In the former pain is less diffused, is felt more at points, and

these are along the course of nerves, and the pain is worse at night when quiet in bed. In the latter pain is diffused over the muscles and aponeuroses and one part is not much more painful than another. The pain is increased on movement, but is diminished by quiet and warmth in bed. The muscle is primarily the seat of the pain which is much increased by passive extension and relieved by pressure. In neuritis there is little pain on extension, but great tenderness to pressure.

*Neuritis and neuralgia.*—Neuralgia is comparatively rare in the extremities. The differentiation must rest chiefly upon the fact that the pain of neuralgia intermits, while that of neuritis is much more persistent. Between the attacks of neuralgia there is not the same tenderness of the nerve trunks, and during the attack the tenderness is more general in its distribution over the area supplied by the nerves. Of course, in neuralgia we never have changes in the muscles due to wasting of the muscle fibres.

*Neuritis and pain of central origin.*—Pains of central origin are nearly always bilateral. They are increased by motion. The diagnosis must depend upon corroborating symptoms.

*Sprain.*—A fixed pain may be the result of sprain or other injury. It should not be forgotten, however, that a slight injury may determine the fixation of a general rheumatic irritation in the injured part. A sudden attack of muscular rheumatism, while making a motion, may be mistaken for a sprain.

*Conclusion.*—The character of pain, although of some importance, is not of great diagnostic value, for the same quality and severity of pain may arise from various causes, and in different individuals the same cause varies almost indefinitely in producing pain.

In questioning a structure to locate a disease we examine it for tenderness of part or the whole of the structure. We test its function to ascertain whether it is impaired or carried on with pain. But structures have more than one function, and when we have indicted an organ we expect to convict it by the additional evidence of the impairment of other functions. Thus in the pain of neuritis we expect usually to find sensation impaired where presided over by the affected nerve.

The best diagnosis is always by exclusion. It is important to bear in mind in the diagnosis of all painful affections that neuritis may be the cause, or that a com-

plicating neuritis may account for part of the symptoms. Most symptoms express themselves through the nervous system, hence the importance to the general practitioner of a knowledge of the peripheral nerves, as it is also necessary for him to appreciate the mental phenomena of the sick. To sum up the points we wish to make in the diagnosis of chronic indefinite painful affections of the extremities—we must approach them with an open mind, realizing that pain may have its origin in the nerve itself, in the tissue to which the nerve is supplied, or it may be of central origin. In our examinations we must not only examine the joints and muscles as a routine practice, but we must examine by palpation the nerve trunks. We must obtain accurate histories because much light will be thrown upon the case by knowing whether the pain has remained stationary in one place or whether it has jumped from one spot to another. A stationary pain is strong evidence of neuritis. Again we must study the distribution of the pain as to whether it corresponds closely to the distribution of some particular nerve.

A recent writer says, "Our tendency to classify cases under various heads will be for the most part inversely proportional to our clinical insight. The difficulty is not to see the resemblance between different classes of cases, but to stipulate the points which differentiate them. It is by this faculty alone that medicine has advanced."

In the preparation of this paper I have consulted the works of Gowers, Starr, Dana, H. C. Wood, Osler, the article on rheumatism in the *Twentieth Century Practice*, and articles in various periodicals.

**Diagnosis of Tubercular Meningitis.**—Deunig (*Centralblatt für innere Medizin*, March 16, 1895). In cases of meningitis where, in spite of all present symptoms, the diagnosis remains in doubt, D. recommends making a lumbar puncture and withdrawing the spinal fluid for bacteriological investigation. In the author's case such a puncture was made, tubercle bacilli in large numbers were found, and successful inoculations practiced on guinea pigs.

FREEMAN.



NOTES ON A CASE OF GENERAL PARESIS WITH  
SPECIAL REFERENCE TO THE STUDY OF  
THE RELATIONS BETWEEN THE TEMPER-  
ATURE, PULSE RATE AND RESPIRATION.

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ABOUT two years and a half ago Drs. Peterson and Langdon made some observations regarding the temperature in cases of general paralysis, and the results of these observations were published in an article which appeared in the JOURNAL OF NERVOUS AND MENTAL DISEASE, November, 1893, entitled, "A Study of the Temperature in Twenty-five Cases of General Paresis of the Insane." The conclusions they arrived at were as follows, viz.:

1. As regards the average bodily temperature, we find it to correspond to physiological normes. The statements of our predecessors as to hyperpyrexia, or subnormal averages cannot be sustained.

2. The diurnal oscillations of temperature in paretics also correspond to physiological normes. The statements to be found in literature as to extraordinary daily variations being frequent in these cases are absolutely erroneous.

3. Asymmetrical axillary differences are so small that they cannot be considered as abnormal and certainly not of any diagnostic significance.

4. When unusual variations occur in general paretics their cause must be sought for in conditions not related to the pathological phenomena of paralytic dementia, but depending upon thermogenic features unrecognized by the physician, or "masked" by the mental state of the patient. Thus in case two of our series an increasing hyperpyrexia was noted during the second week's observation, but the pneumonia causing it was "masked" until the fifth or sixth day, patient dying on the sixth day. Again, in case ten, where the highest single daily oscillation was 3.4°, and the average daily oscillation for the week 2.2°, patient suffered from bed sores which

undoubtedly produced some septicæmia. That variations in temperature may take place in connection with the paralytic and convulsive seizures of these cases, we do not gainsay, but we have made no observations under such circumstances.

I would say here, that I was an assistant at the Hudson River State Hospital at the time these observations were made, and became much interested in the subject, and feel certain that they were made with as much accuracy as possible, although in my opinion, there was not enough discrimination shown in the selection of the cases in order to secure the most trustworthy statistics. Thus all the cases of general paralysis in the male department were observed regardless of whether these cases were in the second or third stage of the malady, or whether they were cases of dementia paralytica or paralytic insanity, accompanied with more or less motor restlessness, or whether they were exposed to circumstances which would cause unnecessary cerebral excitement.

In the case under consideration, I not only made similar observations concerning the temperature, but also went a step further and endeavored to ascertain the relation of the pulse rate and the respiration to the temperature, that is, whether they held a definite ratio to each other, or not.

I wish to state, in passing, that I had a competent nurse make the following records and can vouch for their accuracy. The nurse was directed that the patient should be perfectly quiet for at least twenty minutes before making the records of the temperature, pulse and respiration.

Before proceeding further a brief survey of the patient's previous history may be of interest.

A. B., male, age on admission 45 years; native of United States; single; lungs and heart normal. Contracted syphilis in 1883, and had secondary lesions of the skin. Had been treated for syphilis.

Exciting causes of malady are said to be excessive devotion to business, and sexual excess. Predisposing cause; syphilis. The patient showed symptoms of mental disturbance in the summer of 1890, and in the fall of the same year was seen by an eminent alienist who was of the opinion that paresis would probably develop. Subsequently the patient was taken to Europe, and there subjected to a course of anti-syphilitic treatment, but with-

out obtaining any beneficial results, mentally or physically. The patient was admitted to Greenmont, September 30, 1892, and was found to be in the first stage of dementia paralytica.

In June, 1894, I made some observations concerning the relations between the temperature, pulse rate, and respiration which extended over a period of two weeks, namely, from June 1 to 14, inclusive, the records being made between the hours of 7 A.M. and 9 P.M. daily. The patient, at this time, was well advanced in the second stage of his malady. He was always in a reclining posture when the above named records were made. On consulting the accompanying charts and tables, it will be seen that during the first seven days, although the oscillations of temperature were comparatively slight, the temperature ranged above normal, except in three instances. The greatest diurnal variation during this period was between  $98^{\circ}$  and  $99.4^{\circ}$ , which was noted on the 6th inst. The highest temperature reached during the week was at 7 A.M. on the morning of the 5th inst., which was  $100.3^{\circ}$ . The patient was reported to have slept badly during the night, and to have been very violent during the day.

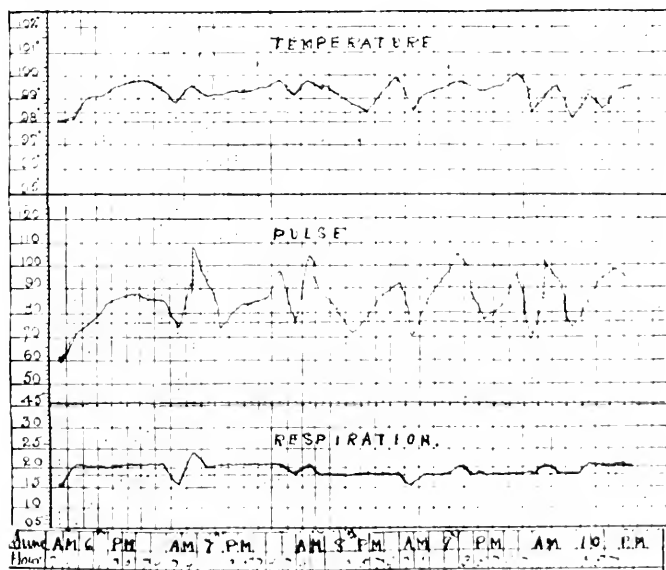


CHART 1. Showing the relation between A. B. S. Temperature, Pulse and Respiration from June 6th to 10th inclusive, 1894.

By a careful examination of the chart and table it will be seen that the ratio between the temperature and pulse is very variable and that they hold no definite relation to each other. The same may be said of the ratio between the pulse and the respiration. The most marked instance of variation from the usual ratio between the temperature, pulse rate and respiration was noted on the 5th inst., at 7 A. M., when the temperature registered  $100\frac{3}{4}$ , the pulse 68 and the respiration 16.

In the chart and table showing the temperature,

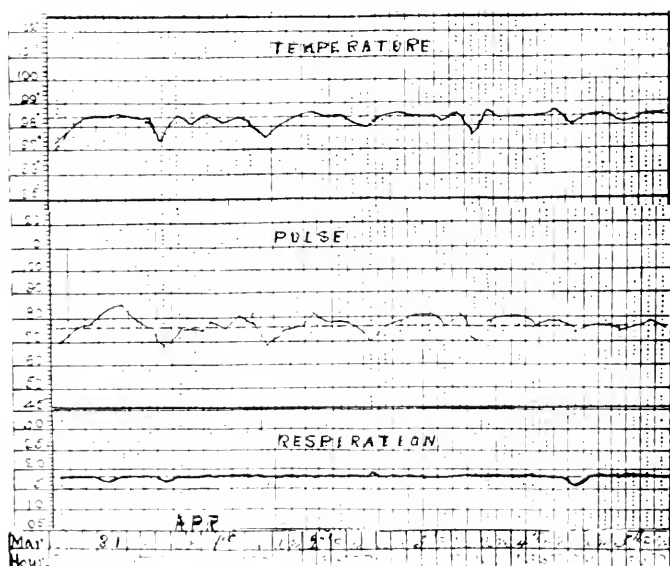


CHART 2. Showing the relation between the nurse's Temperature, Pulse, and Respiration from March 31st to April 5th inclusive, 1895.

pulse rate, and respiration from June 8th to 14th, inclusive, there is nothing of further interest, except that on the 11th inst. at 7 A. M., the temperature registered  $100\frac{3}{4}$ , and patient was reported to have had a slight convulsion lasting for twenty minutes. The greatest diurnal variation during this period was  $1\frac{3}{4}$  which occurred on the 14th inst.

On Feb. 21st, 1895 I renewed my observations of the previous June. The patient was at this time in the third stage of general paralysis and was confined to his

room. During the first few weeks, he sat in a reclining chair for a part of each day, but later on was entirely bed-ridden. The endeavor was made to be as accurate as possible, the records not being made until the patient had remained perfectly quiet in his reclining chair, or bed for at least twenty minutes. Although the pulse and respiration become very variable, even in health on slight provocation, still by making a careful study of the charts and tables covering a series of weeks, it will be seen, that their relation to each other and to the tem-

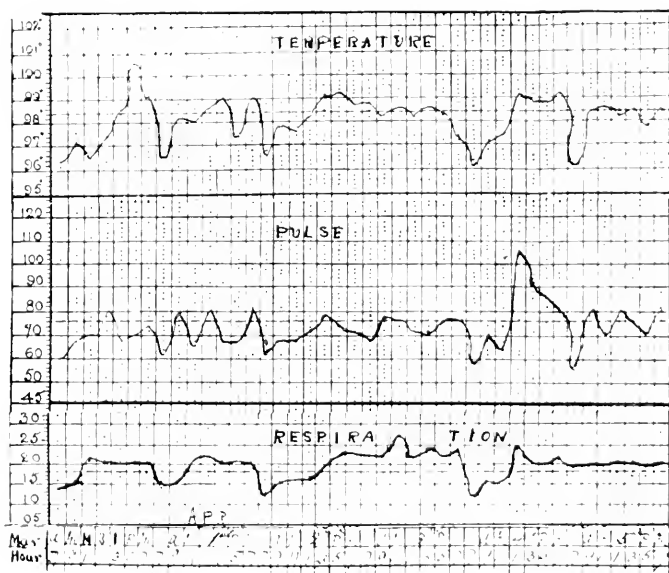


CHART 3. Showing the relation between A. B.'s Temperature, Pulse and Respiration from March 31st to April 5th inclusive, 1895.

perature is more variable than under normal circumstances, the same precautions for accuracy having been taken. Thus from March 31st to April 5th, inclusive, I had the nurse take his own temperature, pulse rate, and respiration at the same hours as he took those of the patient. By comparing the two charts for those dates, a great contrast will be observed.

I also give the records of the convulsions, and the hours at which the most of them occurred, in order that a study may be made between the occurrence of the con-

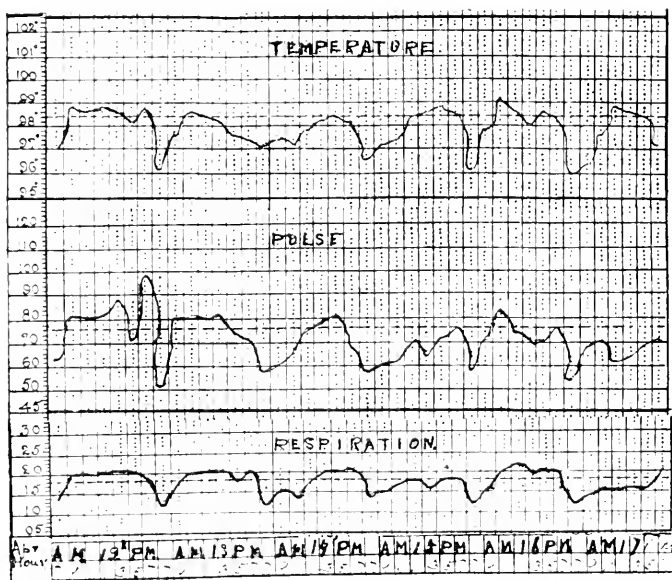


CHART 4. Showing the relation between A. B.'s Temperature, Pulse and Respiration from April 12th to 17th inclusive, 1905.

vulsions and the condition of the bodily temperature. From a study of the present case, these two conditions seem to be entirely independent of each other.

In this case both general and localized convulsions were very frequent. Spasms of one or both eye-lids occurred at times without any other noticeable convulsive movement. Thus on March 13th patient had a spasmodic closure of the right eye-lid lasting for three hours; and the nurse was unable to open it, except by force.

The patient was also subject to spasmodic contractions of the laryngeal muscles, as sometimes occurs in locomotor ataxy, followed by cyanosis, dyspnoea, and cough, lasting at times, four or five minutes. After cessation of the laryngeal spasm, the cough would be quite severe while large quantities of mucus would be secreted and would have to be removed from the fauces by means of a swab, and by spraying the nose and pharynx. One of these attacks is thus described in the daily report. "At 1.10 P. M., my attention was directed

toward patient who was making a gurgling sound in his throat, as though choking. At the same time he was making frantic efforts with his hands as though to pull something out of his throat. He was dark purple in face, and gasping for breath, these symptoms lasting fully five minutes. I raised patient somewhat and lightly tapped him on chest, and after a time the tapping seemed to relieve him somewhat, and then he began to cough; a regular bronchial cough, which lasted twenty minutes. Was very exhausted and unable to articulate for three hours, after which time could only say a word with great difficulty. During these attacks, both before and after, the temperature remained very low."

About a week before death the patient developed a mild attack of bronchitis, and the mucus was secreted so abundantly that the patient became almost asphyxiated thereby, not having strength enough to cough it up. The most efficient means adopted for the relief of this condition was to thoroughly spray the nose and fauces with a warm salt solution. This afforded marked and prompt relief to the patient. The warm salt solution would liquify the mucus collected in the fauces and would also induce coughing, and by placing the patient on the abdomen, the mucus would run copiously from the mouth. The relief thus obtained would often last for an hour, or two. Thus on April 26th the nurse reports having removed as much as the equivalent of five teacupful of mucus during the day.

I here append comparative charts and tables showing the relation that the temperature, pulse rate, and respiration held to each other in the daytime, from Feb. 21st to April 18th, inclusive, and the greatest diurnal variation in temperature in each seven days. The lowest temperature reached is not recorded on these charts. At 3 A. M., on the morning of April 16th, patient had a convulsion. The temperature then registered  $95\frac{1}{2}$ , pulse 70, respiration 31. I found practically no difference between the temperatures on the right and left sides of the body, and thus have made no records concerning them.

On and after April 24th complications developed, and as a result much wider oscillations occurred in the temperature, pulse rate and respiration. The patient died May 1st, 1895.

Again referring to Dr. Peterson's article, he says in

the fourth conclusion, "When unusual variations of temperature occur in general paretics, their cause must be sought for in conditions not related to the pathological phenomena of paralytic dementia, but depending on thermogenic features unrecognized by the physician, or masked by the mental state of the patient."

In the case under consideration no cause, or causes existed to produce the variations in temperature described except those purely attributable to the general paralysis as such. It must be remembered also that from Feb. 21st to April 18th, inclusive, when these observations were made, the patient was confined to his room, which was kept at a uniform temperature and that these observations were made in the spring, when the external thermometric changes were comparatively slight, and were not made in the summer, when the atmospheric temperature is generally high during the twenty-four hours, causing the patient to become restless, irritable, and more susceptible to variations in the bodily temperature, owing to more or less sweating, prolonged diarrhœa, hemorrhages, or general exhaustion.

It would seem to the writer that a great deal of light might be thrown upon the relations of the temperature, pulse rate, and respiration in general paralysis, if a careful study of these cases was made, and at the same time afford a basis for the study of the progressive morbid changes which occur in the sympathetic nervous system.

In order to make an accurate study of these phenomena, the cases to be observed must be very carefully selected. Thus cases of general paralysis in which frequent attacks of motor restlessness occur must necessarily be eliminated, even if these patients are confined to their beds, as the effect of the heart and respiration will be such that the observations will be of no practical value. Each patient should be in a separate room, or a few patients can be associated in a small isolated section of the ward, in order to eliminate as far as possible all external causes of cerebral excitement; and the rooms, or dormitory should be kept at a uniform temperature. The observations will be more accurate if made in the spring, or fall, when the variations in the atmospheric temperature are comparatively slight. The observations must be considered as valueless, if serious complications supervene, such as a severe general bronchitis, pneumonia, profuse, and prolonged diarrhœa, etc., etc. Here and there, in every large hospital for the insane,



cases can be found which are most favorable for study, and these are cases typical of the fundamental form of General Paralysis of the Insane, known as Dementia paralytica, which is so carefully described by Regis. It is far better to select our cases, and extend our observations over a series of years, and have them approximately correct, than to make a large number of observations and run the risk of having the results of our studies untrustworthy.

TABLE SHOWING GREATEST DIURNAL VARIATIONS IN TEMPERATURE FOR EACH WEEK, FROM 7 A. M. TO 9 P. M.

	Degrees.
February 21 to 28th, inclusive,	2 $\frac{1}{2}$ .
March 1 to 7,	" 5 $\frac{1}{2}$ .
March 8 to 14,	" 2 $\frac{1}{2}$ .
March 15 to 21,	" 3 $\frac{1}{2}$ .
March 22 to 28,	" 3 $\frac{1}{2}$ .
March 29 to April 4,	" 3.
April 5 to 11,	" 2 $\frac{1}{2}$ .
April 12 to 18,	" 2 $\frac{1}{2}$ .

TABLE OF CONVULSIONS.

Dates and hours of occurrence of convulsive seizures, from February

21 to 28, inclusive :		
February 21, 1895.	Slight convulsions.	In afternoon.
February 22, "	Severe convulsions lasting for one hour.	3.30 p.m.
February 23, "	Severe convulsions of right side, lasting forty minutes.	In afternoon.
February 24, "	Severe convulsions lasting one-half hour.	3.30 p.m.
February 25, "	Severe laryngeal spasm lasting for five minutes, followed by severe coughing spell.	1. 10 p.m.
	Swallowing often difficult. Often has spasms of either one or both eyelids quite independent of spasms of other parts of the face.	
February 26, "	Laryngeal spasm lasting for five minutes.	2 p.m.
	Slight facial spasm.	5.30 p.m.
	Complains of pain in the head. Keeps hand on head, on left side towards back part.	
February 27, "	General convulsions lasting twenty minutes.	5.10 p.m.

Dates and hours of occurrence of convulsive seizures, from March 1 to 7, inclusive :

March 1, 1895.	Had slight convulsions and two fainting attacks.	During afternoon.
March 2, "	Convulsion of jaw.	7. a.m.
March 6, "	Convulsion of right side of face and of right eyelid.	6.45 a.m.
	Right eyelid closed tight and could not be opened without using force. Spasm continued until 8 a.m.	
	Convulsion of lower limbs.	8.15 a.m.
	Laryngeal spasm.	9.15 a.m.
	Laryngeal spasm.	6.30 p.m.
March 7, "	Convulsions of left side of face.	8.40 a.m.
	General convulsion.	9.30 a.m.
	Convulsion of right arm.	3.30 p.m.
	Lost use of lower limbs from	6 to 8 p.m.

Dates and hours of occurrence of convulsive seizures, from March 8 to 14, inclusive :

March 8, 1895.	Convulsion.	3.30 p.m.
	Fainting attacks at intervals during the day.	
March 9, "	Convulsions.	4 p.m.
March 10, "	Slight facial convulsions.	In morning.
	Laryngeal spasm.	In afternoon.
March 13, "	Spasm of right eyelid lasting for three hours.	In morning.

Dates and hours of occurrence of convulsive seizures, from March 15 to 21, inclusive :

March 15, 1895.	Laryngeal spasm.	1 p.m.
	Facial convulsions.	5.30 p.m.
March 16, "	Severe convulsions.	2.40 p.m.
March 20, "	Convulsions of left side of body and laryngeal spasm.	1 a.m.
March 21, "	Convulsion of right side of face. Right eyelid closed for seven hours.	

Dates and hours of occurrence of convulsive seizures, from March 22 to 27, inclusive :

March 22, 1895	Convulsions and laryngeal spasm.	11 a.m.
March 23, "	Convulsions.	1 p.m.
	Convulsions.	10 p.m.
	Frequent coughing spells.	
March 24, "	Convulsions from	10.30 to 11.15 a.m.
March 26, "	Convulsions from	7 a.m. to 1 p.m.
	Convulsions followed by coughing spell.	10.20 p.m.
	Patient had seventeen convulsions during these twenty-four hours	

March 27, "	Convulsions.	2.30 p.m.
March 28, "	General convulsions.	7 a.m.
	General convulsions.	1 p.m.
	General convulsions followed by laryngeal spasm.	1.30 p.m.

Dates and hours of occurrence of convulsive seizures, from March 29 to April 4, inclusive :

March 29, 1895.	Three attacks of convulsions on left side of face between laryngeal spasm.	9 and 10 a.m. 6.25 p.m.
March 30, "	General convulsions.	1.15 p.m.
March 31, "	Convulsions.	In afternoon.
April 1, "	Convulsions.	During day.
April 3, "	Frequent attacks of convulsions during night. Had several convulsions followed by violent attack of coughing.	3.15 p.m.
April 4, "	Convulsions followed by violent coughing.	1.20 p.m.
	Convulsions.	10 a.m.
	Convulsions.	1 p.m.

Dates and hours of occurrence of convulsive seizures, from April 5 to 11, inclusive :

April 5, 1895.	Frequent laryngeal convulsions.	During night.
April 6, "	Facial convulsions.	7 a.m.
	Facial convulsions.	9 a.m.
April 8, "	Four general convulsive attacks.	During the day.
April 9, "	Convulsions during night, followed by coughing attacks.	
April 10, "	General convulsions.	6.30 p.m.
April 11, "	General convulsions.	2.40 a.m.

Dates and hours of occurrence of convulsive seizures, from April 12 to 18, inclusive :

April 12, 1895.	General convulsions.	7.15 a.m.
April 15, "	General convulsions.	11.10 p.m.
April 16, "	General convulsions.	3 a.m.
	General convulsions.	11.30 a.m.
April 17, "	Convulsions of face.	2.10 p.m.
April 18, "	General tonic spasm.	7 p.m.

## DUPLEX PERSONALITY.—ITS RELATION TO HYPNOTISM AND TO LUCIDITY.<sup>1</sup>

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### ABSTRACT.

**A**FTER a hundred years of obloquy hypnotism is accepted as a fact in mental science, and being so accepted its claim to consideration and an honorable place are two-fold: first, on account of its practical value as a therapeutic agent, and second, on account of its philosophical value as a key to many phenomena which have formerly been rejected altogether, or else have been looked upon as supernatural. Amongst these unusual phenomena two of the most striking and important are duplex or multiplex personality, and lucidity or clairvoyance.

The systematic study of duplex personality is only of very recent date; but material has now been collected which demands careful study. Three cases are selected as typical examples—that of Félicité X., reported by Dr. Azam, of Bordeaux; that of Ansel Bourne, reported by Dr. Hodgson, Secretary of the American Branch of the Society for Psychical Research, and that of Alma Z., reported by the author of the paper, in the *JOURNAL OF NERVOUS AND MENTAL DISEASE* for September, 1893. In each of these cases, from some physical cause, such as sickness, debility or shock, loss of consciousness occurred, and when consciousness returned it was found to be a consciousness altogether different from that which existed before: the patient looked, talked and acted altogether like a different person, and this new consciousness or self claimed no partnership, no kinship and very little acquaintance even with the original personality, but on the contrary, it claimed to be an entirely independent person. In some cases as, for instance, that of Félicité X., the primary self was weak, sickly, melancholy

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<sup>1</sup> Read before the Neurological Section of the American Medical Association at Baltimore, May 7, 1895.

and indolent; while that which came to take its place was in good health, cheerful and industrious. The two states alternated frequently, the second state often continuing for days and even months together. During all this time the primary consciousness was abolished, and when it returned it had no knowledge whatever of what had transpired in the second state, but all the time so occupied was a blank. On the other hand, all the different occasions upon which this second consciousness came into action were linked together in one connected and consistent chain of memory, having related thoughts, the same sentiments, opinions, code of morals, virtues, vices, and the same general character and personal history, but all entirely different and often altogether opposite to the sentiments, personal history and opinions of the original self. It presents all the characteristics of a real and distinct personality, and it has been variously designated as, (1) a different stratum of consciousness; (2) a subliminal self; (3) a second personality.

Having differentiated such a second condition or personality, we look for other cases having analogies to these rarer types, and the first and most obvious is the condition of ordinary somnambulism. Here we have the same general conditions which marked the cases already referred to. First, the primary consciousness is blotted out; second, another consciousness or personality takes its place; third, all the different occasions upon which the second consciousness appears are linked together in one consistent chain of memory, constituting a personality perfectly distinct from the primary one.

Looking still further we find instances where the same conditions are brought about by means of hypnotism. A marked example of this is presented in Dr. Richet's case of Madame B. Here three distinct personalities successively appeared. Madame B., or Léonie, a very modest, dull and common-place French peasant woman, forty-five years of age, being hypnotized, became a flippant, forward, frolicsome young woman whom the professor named Léontine. Upon further hypnotization and a deeper trance there appeared a sedate, very proper and much more intellectual person who was named Léonore. All these personalities were separate and distinct, had their own characteristics and peculiarities with which they were always consistent. Léonie knew nothing of Léontine nor of Léonore. Léontine, the second personality, knew of Léonie who preceded her,

but not of Léonore who came after her. While Léonore knew both Léonie and Léontine—and each was desirous not to be confounded with the others. Ansel Bourne also passed at once from the primary to the second personality by hypnotism, and then related all the incidents which occurred during six weeks spent previously in that condition as A. J. Brown.

We find this second personality coming to the surface under at least these three conditions, namely: physical weakness, sickness or shock—ordinary somnambulism and in the hypnotic trance.

We find this subliminal self active also in sleep when no active somnambulism is present, especially in veridical dreams, and also in reverie or the condition between sleeping and waking when the ordinary consciousness is quiescent; and well authenticated examples are given. In some of these states and especially in somnambulism, reverie and the hypnotic condition we find the subliminal self possessed of unusual or supra-normal perceptive powers—it is able to go away hundreds of miles, see what is transpiring and report it automatically through the physical organization or impress it upon the primary consciousness in various ways. Examples of the exercise of this faculty are given, occurring especially in dreams and in the hypnotic trance.

Alleged facts of this kind are not received by the strictly physiological school of psychologists, but facts are facts, whether received or not, and apart from any theory regarding it, the fact of visual perception gained where vision by the physical organ of sight is impossible, is just as well established as any fact in nature. Cases were reported by Dr. Dufay before the Psychological Society of which Charcot was President at the time. Dr. Backman, of Sweden, found several cases of clairvoyance amongst his own patients; many cases have been carefully reported to the Society for Psychical Research and the writer of the paper himself has had an excellent example within the past year in a patient under his own observation.

The writer draws the following conclusions.

1. That there does exist in some persons and probably in all a subliminal self or second personality which is able to act at a distance from the physical body and which is endowed with perceptive powers exceeding those of the primary self.

2. That clairvoyance is an attribute of the subliminal self—and in that view is a perfectly reasonable and a proper subject of scientific study.

3. That hypnotism is a means of experimentally bringing into action this subliminal self with its added powers of perception.

Without presuming to dogmatize, the writer is certain that a sufficient number of well-observed and authenticated facts have now been accumulated relating to the subjects here treated to demand the candid and careful consideration of medical men; and it was with hope of giving definiteness to such consideration that he ventured to bring the subject before the Association.

**A Case of Agoraphobia.**—Taylor (*N. Y. Medical Journal*, March 30, 1895), reports the case of a man, aged twenty-six years, who consulted him for "nervousness." The attacks were described as "horrible feelings of fear with palpitation of the heart," occurring when about to cross a street or square, or when left alone in any large open space. The first attack came on five years previously when about to set out for a walk over an extensive plain. Patient attributes this to the loss of an old friend who had accompanied him everywhere. He has always been in good health, but has lately suffered from constipation and indigestion, and since puberty has been subject to recurrent attacks of acne. Was treated three years previously for "soft sore," but never had any secondary symptoms. Is temperate, but a heavy consumer of cigarettes, smoking an ounce of Turkish tobacco daily. There are no evidences of organic disease. He is intelligent and well educated, and a clerk by occupation. In this instance the man was haunted by the fear of insanity suggested to him by some indiscreet practitioner. The author reassured him about his fear of insanity; he was advised to take as much exercise as possible without physical or mental fatigue, to fight against the ideas at every opportunity, to prevent constipation and to moderate the use of tobacco. A sea voyage was also suggested. A short time after he was seen again, but his condition was unchanged. He says he feels more confident of overcoming the attacks altogether, a confidence which the author does not share.

FREEMAN.

## Asylum Notes.

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By R. M. PHELPS, M.D.,

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### *A Study of the Hospital Reports.*

*Proportion of Recoveries.*—The recovery rate should certainly be an important matter in an institution, because recoveries should constitute the main, though not, of course, the sole aim of its existence.

But statistics will nevertheless be always unreliable, because each physician making the judgment, will have his own standard. For example, one may decide by a consideration of the condition of the patient upon leaving the hospital; another will insist upon waiting through a period of trial; again one will insist that there is recovery, only if there be an absence of that frequent feebleness of mind so often clearly seen by the trained observer as clinging to the one going out; another will disregard all such defects. Then again, the number of inebriates included, and especially the proportion of acute or chronic patients admitted, are all determining factors. Indeed, the decision, even of the most conscientious, is more like deciding when winter begins and summer ends, than like making a positive statement.

Things being fairly even, however, it would seem possibly true that the tendency of some of the most studious of officers, is to report the least number of recoveries to admissions. Using approximate numbers, Pontiac, Mich., reports (through Drs. Burr and Christian, who have written upon the importance of excluding their perfectly recovered cases), only a little over 11% of recoveries upon admissions for their last period. Reading a little farther, however, we find that they place the unusual proportion of 31% additional as "improved." But this 11% is the lowest in the whole list of eighty reports now before us, with the exception of Willard Asylum, containing chiefly chronic insane.



Next comes Taunton, Mass., with 12%, and Clarinda, Iowa, with 14%, the latter having, however, an overplus of chronic patients. From then on the list reads: Danvers, Mass., 16%; Worcester, Mass., 17%; Rochester, N. Y., 18%; Kankakee, Ills., 18%; Traverse City, Mich., 18%; Westboro Insane Asylum, Mass., 19%; Bolivar, Tenn., 19%.

Beyond this most of hospitals report from 20 to 35% as recovered.

At the other end of the list, however, we find one hospital reporting 66%, one 59%, another 57%, others 48, 47, 46, 45, 38, 38, 37, 36 and 35%.

*Death Rate.*—The proportion of deaths is often noted as for or against a hospital's reputation, and many of these reports take the trouble to explain or comment on their death rate, giving the reasons for its greater or less proportion. For obvious reasons the proportion in this case is not based on the number of admissions, but should be based on the average population. It is a fairly steady rate in hospitals for the insane, all things being considered, and averages from 5 to 8% annually of the population. Nor can the variations be justly argued for or against a hospital, as the variables are so many and so indeterminable. An estimate of the relationship of this proportion to the death rate among a sane population, is also prevented by several considerations, principal among which we may mention the fact that according to some statistics personally gathered, the average age upon admission is about thirty-eight. The expectation of life at this age would, of course, be less than that for all ages combined. Then, too, general paresis is a fatal disease, while acute delirium and other acute insanities have serious prognosis. Senile dementia being often progressive, is then of not much more favorable prognosis.

*Permanency of Physicians' Service.*—The duration of an assistant physician's stay in a hospital will determine very much the value of his work. Our outside critics who settle for life in a place, would not be able to do much that he does, if uprooted each two to five years; especially, moreover, if during all this time it has seemed possible that at any time some political whirlwind will put another man in the superintendency. It is rare to pick up one of these reports without noting toward the end items of the changes among assistant physicians. By some figuring it can be estimated that

about one physician out of five, in this country, stays in the work for five years or more.

Dr. Worcester, *Journal of Insanity*, April, 1895, says: "Under existing arrangements, in most of the hospitals, the position of superintendent is the only one that can be considered satisfactory as a permanency, either in point of honor or emoluments. In many of them either the assistant physicians cannot marry, or, if allowed to do so, cannot live with their family. Their salaries are in most cases comparatively small, and if they do not succeed after a few years in obtaining a superintendency, they are apt to be looked on as failures."

*Tuberculosis Among the Insane.*—Tuberculosis is a very prominent disease among the insane of hospitals, and the subject is often alluded to in these reports. They occasionally advocate "isolation" wards, or buildings for this class of patients. Dr. J. W. Babcock wrote last year quite an exhaustive review of these troubles. He finds, as others before him have found, a percentage of deaths from tuberculosis about 22%, nearly three times that of outside population, and chiefly among the chronic cases. Admitting all the facts as given, and admitting that unhealthy environment and infection are causes, yet it would seem that there are some modifying causes not mentioned. For example, the age of the insane, averaging thirty-eight, would make the proportion greater. The general complete inactivity and personal neglect of the melancholiac and dement, are also very effective causes, and we have found that these inactive cases are quite uniformly the victims. This subject has a firm and broad practical bearing, however, under any argument, and its review by Dr. Babcock is very timely indeed.

*St. Lawrence Hospital Report.*—The report of Dr. P. M. Wise and staff (Ogdensburg, N. Y.) demands especial note. It seems the well modulated, conservative, conscientious and hopeful report of a staff enthusiastic and painstaking in their work. The opinions of the superintendent seem practical and unbiased, and commend themselves as such.

The managing board states: "The board also wishes to evince its most hearty appreciation of the services of Dr. P. M. Wise, the medical superintendent of this hospital. The labor involved in organizing a complete, new hospital service has been herculean, and his efforts to perfect and extend all departments have been untiring."

Dr. Wise in his report maintains that the pathological research in a hospital, and is in close contact with its clinical counterpart is of most value. He maintains a good training school for nurses; that an absolute prohibition of mechanical restraint is as purely nonsensical and sentimental as would be the prohibition of forcible feeding; and yet says further, that he seldom uses restraint, and thinks a nurse better than a protection sheet. In acute delirium he at times favors four nurses, placing two on in day service and two more at night.

Entrance examinations are made, even to blood discs and hæmoglobin. Nurses' full records are kept, etc. This report will doubtless show to many outside of hospital life, how laborious and full a life it is to those who are fully committed to their work. The papers forming an appendix to this report are too extended for comment here.

*Changes of Officers.*—The following is quoted from the report of the superintendent of the Hospital for Insane at Anna, Illinois:

"As is usually incident to a change of State administration, a change of the management of this institution was effected March 6, 1893.

"At your first meeting, held at the hospital on the above day, Dr. E. B. Elrod, the former superintendent, presented his resignation, which you accepted, and at the same meeting you elected me to succeed him, and for which I now wish to thank you.

"Dr. A. B. Beattie, of Redbud, resigned the position of first assistant physician, May 18, 1893, and Dr. R. M. McCall, of Vienna, was appointed. Dr. S. C. Hall, of Omaha, was appointed to the position of physician at Annex, made vacant by the resignation of Dr. M. J. Benson, of Vienna, May 1, 1893. Dr. R. A. Goodner, of Stone Church, was appointed to the position of third assistant physician, made vacant by the resignation of Dr. N. B. Baker, of Cottage Home. Mr. E. Finch resigned the position of clerk, and Mr. J. L. Hammond, of Murphysboro, was appointed; Mr. C. O. Kimball resigned the position of storekeeper, and Mr. W. L. Wiggins, of Johnsboro, was appointed; Miss Emma T. Mace resigned the position of druggist, and Mr. George H. Wood, of Simpson, was appointed; H. E. Wilson resigned the position of engineer, and B. L. Magee, of Cairo, was appointed; Mr. Mark Whitacre resigned the position of farmer, and James Cooper, of Metropolis, was

appointed. During the month of December, 1893, Mr. Cooper received injuries by being thrown from a buggy, from which he died, and in January, 1894, Mr. Z. T. Roddy, of Salem, was appointed to fill the vacancy. W. H. Smart resigned the position of record clerk, and R. E. Vernor, of Tamaroa, was appointed; Miss Mary E. Bell resigned the position of stenographer, and Miss Grace M. Kimball, of Murphysboro, was appointed; Miss Anna E. Steers has been retained as matron; Mr. H. F. Warren and Miss Etta Peak, supervisors, resigned, and J. E. Detrich and Mrs. Harriet E. Liston were appointed. Many other changes in the various departments have been made."

Dr. W. A. Worcester, in a letter to the April number of *Journal of Insanity*, writes in a conservative and dignified manner, taking some exceptions to Dr. Channing's representation of the standing of insane asylum officers. Dr. Worcester maintains that the insane deserve and should have good medical care, "and that any system which so occupies the time and attention of their physicians with other matter, however important as to render them unable to become thoroughly proficient in this profession, leaves something to be desired."

He thinks institution physicians probably have as much time for research and study as outside physicians; but that often the institution "atmosphere is not professionally stimulating." He describes how "assistants come to have the feeling that professional merit does not count for much," and also the frequent lack of instruction to assistants, or work by them. It seems to the doctor "hardly possible that the same person should be the administrative head, and the medical head, and perform both classes of duties satisfactorily." "To be a first-class physician will tax the ability of most men pretty severely."

He describes a medical officer, who shall examine all newly-admitted cases, all cases of serious illness, shall direct and oversee the treatment, shall suggest topics for study and research, inspect notes of cases, conduct post-mortem examinations, give instructions to the less experienced members of the staff, and who shall receive a salary commensurate with the duties.

His plan seems practically to have two approximately equal officers, the medical superintendent, administrative, and a physician at the head of the medical work.

***The Expiscation of Acute Delirium.***—Dr. H. C. Wood (*American Journal of Medical Sciences*, April, 1895), presents under this heading a very instructive article, exhibiting an effort to logically disentangle acute delirium from the acute manias, confusional states, etc., by clinical and etiological data. His last paragraph shows up briefly and fairly his conclusions. It reads as follows:

“The conclusions which are to mind probably, but not firmly established, are, that all manias of an acute type, which are not intoxication neuroses, and are not due to the presence of organisms in the blood, are divisible into two sections. First, mania proper; second, confusional insanity;—and that each of these diseases becomes, when in its most severe form, an acute delirium. Thus, there would be, first an acute mania; that is, mild acute periencephalitis known when in its delirious form as acute delirium; that is, violent, usually fatal periencephalitis; second, confusional insanity without demonstrable lesion, but probably the result of changes in the ganglionic cells themselves, constituting in its severest form an acute delirium, also, without demonstrable lesion, but, in fact, due to an exaggeration of the unknown ganglionic or other alteration present in the confusional insanity.

***A Study of the Degenerative and Destructive Diseases of the Lungs among the Insane.***—This is the title of an article by Dr. H. A. Tomlinson in the *International Medical Magazine* for March, 1895.

He gives a practical and suggestive study of lung lesions, combining clinical study with closely following post-mortem examinations in order to secure the fullest ideas of logical causation. He describes the lesions found as belonging to three classes: First an unresolved broncho-pneumonia, progressing as catarrhal phthisis to a fatal termination, with or without tubercular infection. Second, a “progressive hyperplasia of the parenchyma of the lungs, gradually destroying function of the organ by encroachment upon the vesicular area and lumen of the bronchioles.” Third, arterial sclerosis, presenting a rigid, inelastic tissue with apparent fibroid increase, although the lungs may be actually much shrunken in mass.

These two latter forms, he says, are commonly infected with tubercle, though not essentially so, and the

hospital conditions favor this infection. He outlines some typical cases to illustrate these points.

The hyperplastic form, he finds the most common, occurring generally between the ages of twenty-five and forty, and caused generally by habits, and position of the patient. The lesions are more commonly in the middle lobe. The interstitial form represents atrophy from impaired nutrition, due to arterial sclerosis, and is found after middle life. The author believes in the claim that there is phthisis without tuberculosis.

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**On the State of the Reflexes After Cross-section of the Cord.**—Gerhardt (*Zeitschr. f. Nervenheilk.*, Vol. vi., Part 1 and 2, 1894.

It was only quite recently that neurologists began to doubt the correctness of the familiar statement: that a cross-section of the upper parts of the cord is constantly accompanied by an increase of the deep reflexes.

Bastian was the first to point out the falsity of this statement; observations corroborating Bastian's views have followed. Bruns, after having given the matter a more careful study, came to the conclusion, that a perfect cross-section of the upper portions of the cord is followed by a diminution or even absence of the reflexes, superficial as well as deep. This is the more remarkable, as the author just mentioned has failed to find any disease whatsoever in the reflex arc or in the peripheral nerves, in spite of a most careful microscopical examination. Bruns explains this occurrence with the aid of Jackson's theory, that the activity of the reflexes are independent of the integrity of the conduction from the cerebellum to anterior cells of the cord.

The author reports a case of a compression myelitis, produced by an angioma of the vertebral bones. The patient presented the familiar symptoms of a compression paraplegia, with partial paralysis of the sphincters. The deep reflexes were increased in the earlier periods of the disease and diminishing with the progress of the disease. They were absent for the last two years. The skin reflexes were increased all the time, but only some, *i. e.*, reflexes producing a flexion of the thigh or the whole lower extremity were increased; all the others were absent.

The microscopical examination revealed a cicatricial tissue taking the place of the cord in an area of about three vertebrae, without a trace of a nerve fibre within.

To the author's mind the theory of Jackson's does not explain this phenomenon, the reflexes having been already absent before the interruption of the conduction in the cord has been perfect. He is more inclined to believe that the pathological process acts in these cases as an irritant to the inhibition leading to paralysis and not to irritation.

FRAENKEL.

## Periscope.

### PATHOLOGICAL.

**A Case of Subacute Nuclear Ophthalmoplegia and Paralysis of the Extremities with Post-mortem Findings.**—Kalischier (*Zeitschr. f. Nervenheilk.*, Vol. vi, pt. 3 and 4, 1895).

Male, 64 years old, of good family and personal history developed without apparent cause (except possibly sorrow, anxiety and misuse of tobacco); first, a right side ophthalmoplegia with associated diplopia and soon after a similar condition on the left side. In a few days the external eye-muscles became paralytic, while the internal muscles which in the beginning were entirely free became paretic. General disturbances, such as fever, headache, optic neuritis, vomiting, vertigo, stupidity, etc., were entirely absent. The other cranial nerves were unimpaired, except for a slight weakness in the internal portion of the right facial. Nearly the same time, perhaps two or three weeks later, there developed a facial symmetrical paresis which went on to paralysis of the extremities, first the upper then the lower, and later the muscles of the buttocks. The extensors were involved more severely than the flexors, the distal ends of the extremities a little more than the proximal ends. Accompanying this there was loss of the tendon reflexes without ataxia and without loss of sense of position. Faradic irritability of the muscles and nerves was diminished, but there was not entire reaction of degeneration. No fibrillary twitchings and likewise no real atrophy of the paralyzed muscles during the course of the disease. Sphincters were intact and no disturbances of sensibility could be made out, although there was slight sensitiveness over the trunks of the cranial and spinal nerves in their peripheral distribution when they were subjected to pressure and also light paresthesia of the hands. After a period of progression all the symptoms became stationary and there was in general a real remission. Especially after rest and in the morning the paralysis would be much improved, but a condition of exhaustion and fatigue would soon replace this. Four and one-half months after the beginning of the disease death occurred rather suddenly from respiratory paralysis without the previous occurrence of symptoms pointing to involvement of the medulla. Power of speech, of swallowing and the intellectual faculties had been undisturbed. Autopsy made twenty-two hours after death. Adhesion of the dura to the skull, especially over right frontal lobes. Over the pia there was some grayish white fluid which was especially noticeable over the convexity of the right hemisphere. Pacchionian bodies extremely large. No evidence of arterial degeneration at the base of brain. Otherwise brain and cord normal microscopically. Microscopical examination showed normal optic chiasm, optic nerves anterior to this, and nerves going to supply other eye muscles. No increase in the interstitial tissue in the nuclei of these nerves. Peripheral nerves and pieces of muscles, which had been removed, were found normal.

In dorsal spinal cord methyl blue stains show the anterior horn cells diffusely and blackly stained, the unformed ground substance is not sharply differentiated from the surrounding chromatic little wands, the

nuclei is easily differentiated from the cells, but the body of the nucleus is very deeply stained. All the cells are poor in processes. The anterior lateral group is scant throughout. The changes in the cells are more marked in the lower portion of the dorsal cord than in the higher. Here the cells are in part pale and colorless, poor in chromatic substance and devoid of processes. Pieces of the cord hardened in the chrome salts and stained with aniline dyes show in the cervical cord a sinking in of the anterior horns with numerous little holes which are apparent to the naked eye, representing the post-mortem dropping out of necrotic atrophic tissues. The ground work of the anterior horns is rarefied, the ganglionic cells are lessened in number and amorphous, poor in processes and diffusely stained. The central, the medial and the anterior are more involved than the posterior lateral, although the defect would seem to be more in the posterior portion of the anterior horns. The right anterior horn is smaller and more pointed than the left. Below this some blood cells are found lying free in the tissues. The blood-vessels walls are not much changed.

The extra medullary anterior and posterior roots in the cervical region are extremely degenerated.

Throughout the entire cord in the gray matter were found in places dilatation of the blood-vessels and small hæmorrhages. No degeneration of the white matter except a very slight evidence of involvement of the columns of Goll and the part of the pyramidal column bordering on the gray matter. Pia mater, especially in cervical region, thickened at posterior part of cord.

Same condition of small hæmorrhages in the motor nuclei of the eye muscles in the vicinity of the aqueduct of Sylvius and a similar but very much slighter condition in the motor nuclei of the medulla. The sensory nuclei of the medulla were almost without exception not at all encroached upon. The right facial nucleus at its inferior end was less well developed than the left. At this level the bottom of the fourth ventricle is filled with innumerable blood-vessels, little holes and small clumps of blood. Higher up are seen small hæmorrhages laterally in the side of the floor of the fourth ventricle and the aqueduct. Ventrally to the posterior long bundle and mesially to the substantia ferruginea at this height was a large hæmorrhage.

J. C.

**Further Contributions to the Study of Spinal Disease Occurring in the Course of Pernicious Anæmia.**—Nonne (*Deutsche Zeitschrift für Nervenheilkunde*, Bd. vi., p. 313).

The author, who had already contributed a valuable study of the changes found in the spinal cord in two cases of pernicious anæmia to the literature of the subject records in the present paper the results of the very thorough microscopic examinations of seventeen new cases of pernicious anæmia. A brief clinical history, a careful report of the physical condition prior to death, and a detailed account of the gross and microscopic changes found in the various organs, especially in the spinal cord, are given in each of the seventeen cases. The spinal cord in each case was scrutinized very thoroughly in order to detect the very slightest changes and to study the morbid processes in their very incipency. From an anatomical point of view Nonne distinguishes three groups amongst his seventeen cases. In the seven cases in the first group no changes whatever or changes so slight as hardly to justify their being termed morbid were found; in some of these cases minute changes, such as swelling of a number of axis cylinders and their sheaths were noted in the region of the middle root zones. The author is inclined to regard these changes as the very first signs of disturbed nutrition in the affected area, although no positive statement to that effect can be made as yet.

The second group consists of three cases showing beyond a doubt the characteristic spinal lesions in the early stages: a few small and



even minute foci of acute degeneration were found in the white substance of the cord, always in the immediate vicinity of a blood-vessel.

In the seven cases forming the third group the spinal disease was present in more or less advanced stages. In the cervical cord the foci of degeneration were most numerous, they involved the posterior and middle root zones most commonly, and were arranged more or less symmetrically on the two sides of the cord; the columns of Goll are degenerated throughout. Irregular foci of degeneration were also present in the lateral columns without being confined to any special system. In the centre of every focus of degeneration a small diseased blood-vessel could almost invariably be made out. The process in the dorsal cord resembled that in the cervical portion, but was less marked. The lumbar region showed but very slight, if any, changes. (Case 11, Ficht.)

In discussing the morbid anatomical conditions found the author emphasizes the important bearing of the diseased blood-vessels to the foci of degeneration. The morbid process in the blood-vessels effect primarily the peri-vascular lymph-spaces, which are often clogged with peculiar hyaline masses; subsequently the adventitia and media are involved in the morbid process and become thickened, whereas the intima seems to be affected last of all.

The study of the early stages of the spinal disease demonstrates the important fact, that it begins in distinct foci of degeneration occurring at some distance from each other; in the course of the disease these foci spread and at the same time new foci develop, which may unite with the older ones, so that the cord is finally involved quite extensively. The disease begins invariably in the cervical cord, and in its later stages the latter is without exception affected more extensively and intensely than the lower portions of the cord.

A marked predilection is shown by the disease for the middle root zones, other parts of the posterior column are, however, also often involved and even lateral and anterior columns may contain foci of degeneration at an early date. Numerous foci in the lateral and posterior columns may by uniting with each other or by causing secondary degeneration simulate the conditions of a combined systemic sclerosis.

A very important point recognized already by Lichtheim and Minnich and confirmed by the author, is the fact that the disease attacks the white substance of the cord exclusively, the posterior roots and the gray columns are never involved.

The author compares at length the changes found in the cord in this class of cases with those found in ergot of rye poisoning (Tuczek), in pellagra, disseminated sclerosis (Popoff's recent studies), paralysis agitans (Redlich), syphilitic myelitis, hereditary chorea, and chronic alcoholism, showing both the points in common in the more minute histological changes found in these various diseases and the distinctive features of each disease, especially as compared with the lesions occasioned by the disease under discussion.

In three cases in which peripheral nerves were examined, no changes were found in any of them. Certain clinical symptoms occurring in the course of pernicious anemia point to disturbance in the nutrition of the brain; the study of the spinal lesions induced by the disease being practically completed by the author's investigations, it is to be expected that investigators will turn their attention now to a study of the lesions in the brain.

In comparing the nervous phenomena observed during life in his cases with the changes found in the cord the author remarks that in its early stages the spinal disease rarely gives rise to any distinct symptoms and in its later stages the latter are not nearly so pronounced as in locomotor ataxy, in ergot of rye poisoning and in pellagra, even in the presence of extensive lesions. This is accounted for partly by the fact that the disease predominates in the cervical region, which gives rise to less

marked symptoms than the lesions of tabes, etc., in the lumbar region. The clinical symptoms most frequently observed are, loss of the knee-jerks, pains, paresthesia, ataxy, muscular weakness. A table of the seventeen cases investigated concludes this excellent paper, which well deserves being read in the original. STEGLITZ.

**Anatomical Contribution to the Study of Combined Systemic Diseases of the Cord.**—Jakob (*Zeitschr. f. Nervenheilk.*, Vol. 6, Part 1 and 2, 1894).

A report of the anatomical examination of a cord found accidentally amongst the specimens of the pathological laboratory in Erlangen. The clinical history of the case could not be obtained. The cord had been preserved for years in alcohol of 70%, after having been hardened in Müller's. The staining was done after Weigert-Pál, with neutral carmine, alum-carmine, and hæmatoxyein-eosin.

The examination showed: 1. Degeneration of the direct and crossed pyramidal tracts; the former up to the level of the upper lumbar region, the latter as far down as the conus. The intensity of the degeneration has been found to increase from above downwards. 2. Total degeneration of the cerebellar tracts in the whole length. 3. Incomplete degeneration of the columns of Goll. 4. Incomplete degeneration of the columns of Burdach. 5. The anterior and lateral roots are normal; the lateral and posterior root zones degenerated here and there. 6. Anterior horns normal, except a slight diminution of the fibres taking a transversal course. Clark's cells normal; anterior roots normal; posterior roots show some disease. The degeneration of the posterior columns does not reach so far down as the degeneration of the lateral columns. All degenerated areas are symmetrically distributed on the cross section of the cord.

These findings lead the author to the following conclusions:

The symmetry of the diseased areas, their anatomical course and the absence of a primary focus in the whole extension of the cord, justify us in regarding combined systemic degenerations as a disease originating primarily in the nervous, and not in the accessory tissue of the central nervous system, *i. e.*, the disease starts in the nerve fibres proper and not in the vessels, neuralgia or meninges of the cord. The absence of a distinct myelitic focus militates against the theory of it being a focal or diffuse myelitis.

The symmetric distribution of the diseased areas is the expression of the elective regularity of the process, following the same path as the embryonic development of medullary sheaths to the fibres. Both ways the function of the fibres seems to have some importance; fibres of similar or allied function getting attacked by the disease and getting simultaneously medullated.

The histological nature of the process has been difficult to ascertain because of the fact that the cord was so long in alcohol. The signs of absolute inflammation found in the environments of the degenerated foci are secondary. The increased demand exercised on the resting fibres during the development of the disease might produce the dilatation of the vessels and the removal of the products of degeneration can be looked upon as a sort of mechanical or chemical stimulus. Noteworthy is the absence of disease in Clark's cells in spite of the total degeneration in the cerebellar tracts. FRAENKEL.

#### CLINICAL.

**The Disturbances of Sensibility of the Globe of the Eye, and its Appendages in Locomotor Ataxia.**—Berger (*La Méd. Mod.*, November 2, 1894). The disturbances of sensibility of the skin in locomotor ataxia are well known, but those of the visual sphere seem to have escaped the notice of the authors up to the present time. In the

area of distribution of the trigeminal nerve, some importance, diagnostically, has been attached to anaesthesia of the pharynx, and peri-orbital anaesthesia of the skin, which, latter, is found in certain cases of tabic atrophy of the optic nerve. The following cases have been observed by the author: Observation I. Woman, 52 years old, had three delicate children and one miscarriage; no history of lues, but had periostitis of the cranium, cured by K. I. Present condition.—Symptoms of tabes well marked, diplopia, and paresis of the accommodation muscles. Tactile anaesthesia of the inferior palpebral and bulbar conjunctiva. Observation II: Man, 46 years old, weak constitution. No history of nervous disorders or lues. Has a healthy daughter. Condition present in March, 1894—general paralysis, complicated with tabic symptoms. August 10, 1894—Mydriasis of left pupil; reaction to light and accommodation present in the right eye; in the left, the reaction to light is absent, to accommodation present (unilateral Argyll-Robertson's sign). Atrophy of the optic nerve; no Romberg's sign; knee-jerk absent on the left side; diminution of sensibility of the cornea in certain places; impaired localization of tactile sensibility over the skin of the nose, cheek, temples and infra-orbital region. Observation III: Strong man, 72 years old. Had gonorrhoea several times, no syphilis. For twenty years suffered from shooting pains. August 10, 1894—No knee-jerk on the right side; on the left, diminished; bilateral myosis; Argyll-Robertson's sign present; acuity of vision diminished in both eyes; optic atrophy. On the right side, anaesthesia of the inferior palpebral and bulbar conjunctiva; impaired localization over the skin of the upper lid, the forehead, and the temples. On the left side anaesthesia, of the temporal portion of the inferior palpebral conjunctiva; defective localization of the skin over the temporal region of the forehead and cheek. Observation IV. Weak man, 55 years old, with hereditary neurotic taint. In 1871 had indurated chancre, followed by an erythematous eruption; underwent an anti-luetic treatment. Was well until 1887, then it was observed that his character began to change, he becoming very impressionable. Since had headaches; and in 1891 sciatica, disagreeable sensations in and around the eyes, and atrophy of the optic nerve. Was accustomed to smoke fifteen to eighteen cigars daily. July 24, 1894—Romberg's and Westphal's sign present on both sides (two years previously the latter was only unilateral), tremor of the hands and tongue, convergent strabismus (due to paralysis of the sixth nerve on the right side and paresis of the same on the left); never had diplopia; bilateral myosis; Argyll-Robertson's sign; visual field diminished; anaesthesia of the palpebral conjunctiva, and diminution of the corneal sensibility (no reflex on touching the cornea); impaired localization of tactile sensations over the skin of the lids, temples, forehead, and cheeks. A peri-orbital gunmata, 1 to 2 cm. in width, is anæsthetic to the touch. Observation V: Weak man, 33 years old. Suffered from general nervousness and insomnia, following the application of cocaine for operation in the nose. In 1892 Professor Haab, in Zürich, detected atrophy of the optic nerve. Condition present August 4, 1894—Bilateral diminution of the visual field; atrophy of the optic nerve; anaesthesia of the palpebral and bulbar conjunctiva of both eyes; perceptions of heat and cold absent; impaired localization of sensations in the upper part of conjunctiva, and over the skin of the temples. Conclusions: In locomotor ataxia various troubles may develop in the eye and its appendages. Thus, in the cornea there are a diminution of sensibility, and disturbances of localization of tactile sensation. In the conjunctiva there is partial anaesthesia to the touch and temperature, also impaired localization. The skin of the lids may show disturbances of localization. The surrounding parts of the eye may also be impaired, there being present, peri-orbital anaesthesia, disturbances of localization over the skin of the forehead, temples, cheek, and nose. Summary: 1. There is no relation between the symptoms, above enu-

merated, and atrophy of the optic nerve of the tabic origin, there being cases in which the latter is present, with no disturbances of sensibility of the eye-ball and surrounding parts. On the other hand, these disturbances may be present in a tabetic subject without the co-existence of optic atrophy. 2. The presence of the disturbances of sensibility and localization, above enumerated, found in a case of atrophy of the optic nerve, point to a tabetic origin of the atrophy. MACALASTER.

**Cerebellar Heredo-Ataxia.**—Under the above head, Dr. Pierre Marie has published two papers in French medical journals; he first described and named the morbid syndrome, after putting together and interpreting several observations of his own and of Fraser, Sanger, Brown, Nonne, and Kleppel and Durante.

Paul Londe has recently recorded two cases of the same disease, collected few new observations, and published on the subject a monograph of 250 pages, "*Heredo-Ataxic Cerebelleuse*, Paris, 1895." The disease is a hereditary and family disease, one or more of the ascendants being neurotic and ataxic; more than one of a family also being affected in the same way. It is an ataxic disease, being attended with inco-ordination of movements and a staggering gait. It is also believed to be a cerebellar disease, the ataxia and inco-ordination being referred to a definite lesion of the lesser brain, viz., atrophy of the cerebellar cortex, or more particularly, of the cells of Purkinje. There have, however, been too few well-conducted autopsies to warrant very positive conclusions, though enforced by physiological considerations. The following observation of Londe and Brissaud brings out the salient points of this morbid syndrome: Patient, German, 24 years old; married; is of a neurotic family; died with some nervous diseases attended with trembling. Patient's brothers and sisters all "nervous; one is affected like her. For a year patient has had some difficulty in walking, especially in the dark. On presenting herself at the clinic it was noticed that she staggers.

Walks with head bent forward to see where to place her feet. A swinging pelvic gait (*elle marche du bassin*).

In the standing posture the oscillations of the trunk are constant; not increased by shutting eyes. With eyes open patient cannot stand on one foot. The head oscillates in rotation or flexion, especially when patient is under strong emotion. The speech is thick, jerky, a monotone. Exaggerated buccal contractions (as at angles of mouth) during articulation; no trembling of tongue; nystagnus when patient is looking intently; general inco-ordination, even in arms, which are agitated with choreiform movements when she is standing or walking; shoulder jerks; cannot with eyes open touch tip of nose with finger; some intentional trembling of hands; cannot do needle work or other fine work; muscular sense is intact; realizes position of limbs; has sense of weight of objects, etc., but motion of equilibrium is lost. When she walks it seems to her that she is "too light," that her feet cannot support the body. Instinctively clings to something to support her. Floor seems soft, and gives way under her.

Patellar and wrist reflexes normal; plantar reflex *nil* to tickling; contact is perceived. There is a double dorso-lumbar curvature of the spine.

Negative signs.—Absence of the Argyll-Robertson sign; absence of paresis of ocular muscles; absence of contractures; absence of vertigo and diplopia; absence of fulgurating pains; no difficulty of deglutition; no trouble with sphincters; no visual troubles; intellect only a little weakened; patient is easily fatigued.

She has been rheumatic and hysterical (*grandes crises* twice).

The vertebral deviation and the ataxia began with the pregnancy and accouchment in 1891; from that date were first noted the staggering gait, the oscillations of the head, the tremblings of the hands, etc.

Patient has been treated by suspension, baths, nitrate of silver, "point de feu" along the vertebral column, but without any benefit.

The following case is given by Fraser: A. K., newspaper carrier, aged 30 years. When seen for the first time by the author, in 1869, he staggered like a drunken person. *Debut* in childhood; disease came on gradually. First symptom noticed, an uncertainty in the gait. At the age of seven years the staggering became more persistent; child was taken from school. In 1875 an attack of typhoid fever, from which made good recovery. In 1876, was obliged to abandon occupation; would often fall headlong when about his work; had to lean against objects for support.

Typical appearance.—Marked titubation; body bent forward; head thrown backward; oscillates continually, as though it was too heavy. The eyeballs are the seat of oscillation without true nystagmus; generally turned up and to one side; internal strabismus of right eye. articulation of words slow, guttural, hesitating. When in a sitting position, no trembling nor choreiform movements, except of the head; intentional trembling when he attempts to write; has some difficulty in touching the tip of his nose when eyes are shut; Romberg sign absent. Patient died of tuberculosis June 11, 1879.

Autopsy.—Pulmonary phthisis, cavities; pneumothorax; liver, kidneys, spleen amyloid; cerbrum and cord normal; the cerebellum is very small, and is only half the normal size, 81 grammes instead of 160 grammes. Section shows the white substance apparently normal, but gray matter diminished in thickness; deep vacuoles in the fissures, and representing little cysts of the pia mater; Purkinje's cells diminished in number, extending to less than the normal depth; nuclei absent.

A sister, aged 24 years, of the above patient, is similarly affected. The same clinical picture; onset in childhood; can no longer write; difficulty in walking; is afraid that she will fall on her face; the act of reading is very fatiguing; for four years has not dared to go out alone; eyes oscillate; fixation painful; internal strabismus of right eye during the convergence of the eyes; pupils sluggish; no achromatopsia; speech slow, hesitating; no mental trouble. On lying down, when she places her head on a horizontal plane, she has vertigo, says that there are flashes in her eyes, and that she can no longer see the clock before her. At this moment she does not dare to turn her head and has a sensation of nausea.

Ophthalmoscopic examination of both patients by Dr. Read. Papillæ pale, irregular in their contour, with veins engorged in certain points; arteries contracted; opaque and thick aspect of the choroid.

Family and hereditary data.—The father healthy, but given to drink; a maternal aunt feeble-minded; nine children, five are dead, two of convulsions, one of phthisis; the brother above mentioned was the last; one of the living sisters, married, had convulsions in childhood, and has lost an infant with meningitis.

To sum up: These two cases present a sufficiently clear clinical picture. Inco-ordination of upper and lower extremities, dating from infancy; paresis of right external rectus; commencement of papillary atrophy; troubles of speech; instability of the eyeball, without true nystagmus; staggering gait, and propensity to fall forward; atrophy of cerebellum; diminution in thickness of the gray substance with cysts of the pia mater; diminution in number of the cells of Purkinje; alteration of these structures in form, and of the surrounding stroma.

HURD.

### **Cerebral Hemorrhage as a Sequel of Enterocolitis.**

Emerson (*Medical Record*, April 20, 1895), reports the case of a child, five months old, who had been fed for six weeks on sterilized milk and most of that time had suffered from more or less diarrhoea. After this the food was changed several times, but the stools continued loose, and

at one time were streaked with mucus and a little blood. There was also a slight daily fever with evening exacerbation. Under treatment the child seemed to be improving, when suddenly it became hemiplegic on the left side. Two hours and a half later it commenced to move in the entire left side, but a second attack followed and terminated fatally. The author says that the literature on hemiplegia as a sequel to enterocolitis is very meagre. Keating finds in eighty-three cases of hemiplegia, one due to this cause, but taking the element of scurvy into consideration, we would expect a hæmorrhagic effusion into any of the closed cavities as a result of malnutrition and a weakening of the vascular walls. Two such cases were reported during the summer of 1894. This the author believes to have been the case here, and that the child's milk was impaired in its digestibility by sterilization, whether by converting the soluble proteids into insoluble, by straying the ferment or by emulsifying the fat, he does not know, but considers that a complex fluid as unstable as milk cannot be subjected to continuous heat without detriment to its nutritive properties, except as a temporary measure.

FREEMAN.

**Puerperal Neuritis.**—Bernhardt (*Centralblatt für innere Medizin*, March 9, 1895). A woman, twenty-nine years old, experienced pain in the right shoulder immediately after her third parturition. This was followed by emaciation of the hand, especially of the interossei muscles and ball of the thumb. At the same time numbness of the little finger and the ulnar side of the forearm appeared. Adduction and abduction of the fingers and movements of the thumb were very feeble. In the course of the median and ulnar nerves, the middle form of the reaction of degeneration was found.

In a second patient a neuritis of the left peroneal developed. In a third case, a woman thirty-one years old claimed to have suffered pain in the right shoulder since her eighteenth year. After her first confinement the pain involved the entire right arm, but again disappeared. After the birth of her last child she was said to have suffered for four months, with fever and a bloody discharge, and from this time dates her present complaints. An investigation revealed a neuritis of the right median or ulnar nerves with moderate form of the reaction of degeneration and atrophy of the affected muscles.

FREEMAN.

**Contribution to the Experimental Study of the Nervous Manifestations of Chronic Arsenical Poisoning.**—Beco (*Centralblatt für innere Medizin*, April 6, 1895). Two guinea pigs, three rabbits and two dogs were given subcutaneous injections of Fowler's solution in increasing doses. The two guinea pigs lived eight and fifteen days, the three rabbits one, one and one-half and two months, the two dogs two and three months. The symptoms observed were a conjunctivitis, trophic changes in the skin, and increasing loss of strength. Death took place with progressive cachexia. The spinal cord and nerves, and the cutaneous and muscular nerves were found intact.

FREEMAN.

**Bulbar Manifestations in Syringomyelia.**—Raymond (*Gazette des Hôpitaux*, March 19, 1895). Bulbar manifestations are quite frequent in the course of syringomyelia. It is more rare for this disease to begin with bulbar manifestations. Exceptionally these assume the form of an apoplectiform attack. The observation of Cohen, an American author, is unique of its kind. It appears that syringomyelia can assume the pure bulbar form. In Cohen's case the symptoms were, ptosis on the right side, inequality of the pupils and an inferior bilateral facial paralysis. Naturally the bulbar manifestations of syringomyelia may be associated with all the clinical varieties of this disease.

FREEMAN.

**Exhibition of Three Microcephalic Children.**—Laborde (*La Méd. Mod.*, 5 Année, No. 95), presented before the Academy of

Medicine three microcephalic children of the same family. The eldest, a girl, was twelve years old, the other two were boys, aged ten and eight years, respectively. The parents were healthy, and four years subsequent to the birth of the last mentioned son, two perfectly normal children were born. The afflicted children are in constant motion, scarcely sleeping a couple of hours at night. The wrist drops, the arms are held flexed, and the hands pronated. At times they walk on all fours. The head of the girl measures 35 cm. in circumference, the boys 38 and 29 cm. respectively. All three are idiotic, but the degree of idiocy is less and less marked from the eldest to the youngest. They cannot talk, only uttering at times incomprehensible, reflex cries. They cannot fix their attention, but see and hear tolerably well. They notice acute sounds, and seem to enjoy musical tones. They grasp and play with everything within their reach, but are even more clumsy than monkeys. They do not recognize anybody, not even the people that care for them, and cannot eat alone. They do not hold the urine or feces.

MACALASTER.

**A Case of *Tabes Incipiens*.**—Weil (*Archiv f. Psychiatrie und Nervenheilk.*, Vol. xxvi., p. 745).

Patient had been infected with syphilis when twenty-six years old. Eleven years later had an attack of left side hemiplegia associated with a prolonged attack of unconsciousness. Following this the symptoms of *tabes* developed in characteristic fashion, except for the absence of lancinating pains and with disturbance of the eye muscles. The patient died three years later from phthisis. There had been no particular disturbance of the function of the bladder or rectum and the disease had followed closely the hemiplegia.

Examination of the cord showed degeneration of the left crossed pyramidal tract and of the right direct pyramidal tract, the result of an ancient thrombosed area the size of a cherry in the internal capsule. In certain portions there were changes in the left lateral horn, doubtful in left anterior horn.

The important changes were in the posterior columns. The degeneration is distributed in an irregular manner and does not corroborate the findings of other authors who have written on the subject of *tabes incipiens*. In the lumbar cord it is not the area of middle root zones that is involved but the larger and greater area and which reaches to the posterior median septum. In the dorsal region the degeneration is more unsymmetrical. Only in the cervical region are the changes similar to those found ordinarily in *tabes incipiens*, viz., degeneration of Goll's columns and that portion of Burdach's column bordering on the latter.

J. C.

**Pseudo *Tabes Mercurialis*.**—Gilbert (*Deutsche Med. Wochenschr.*, November 1, 1894), reports a case resembling that of Leyden's (*ibid.* 1893, No. 31, p. 733). The patient was 26 years old, formerly an officer, and had syphilis in 1892, for which he had undergone several antiluetic cures. During the following summer, after being treated with bichloride injections, and K I., he had severe pains, with total paralysis of the lower extremities and muscular atrophy. The large nerve trunks were sensitive, there was reaction of degeneration, and the knee-jerk was absent, but no atactic symptoms. This condition improved under mercurial inunctions, and later K I. and massage, so that the patient was able to walk with the aid of two sticks. The diagnosis of polyneuritis syphilitica was made. Later symptoms of ataxia developed; the gait was uncertain, Romberg's phenomenon present, and diminution of sensibility from the upper third of the thigh downward. The function of the bladder was impaired and the patient complained of burning pains in the upper and lower extremities, dead feeling in the feet, and weakness in walking. After six weeks' treatment with baths, massage, electricity and tonics the objective and subjective symptoms all disappeared entirely. The following spring the patient died of heart failure after an excess of

*vino*. At the same time there were syphilitic ulcers in the throat and on the chest, and probably some syphilitic affection of the heart muscle.

MACALASTER.

**Half-sided Vasomotor Disturbance of Cerebral Origin.**—Kaiser (*Neurol. Centb.*, May 15, 1895).

The case has its interest in the fact that vasomotor disturbances of one side of the body were found associated with localized disease of the brain.

A demented female, 69 years old, in whom insanity followed a blow on the head. There was great weakness of the entire muscular system, no special paralysis. Reflexes all diminished, sensibility blunted. In January, this year, right hand was swollen and red, no change in appearance of left hand. It was seen also that these vasomotor disturbances consisted of a paresis of the blood-vessels throughout the entire right half of the body with the exception of the head. The right foot and hand were most involved. Temperature in right axilla was always  $0.1^{\circ}$ ,  $0.15^{\circ}$  Celsius higher than in the left. After a few weeks patient died suddenly. In the left gyrus supramarginalis was found a sinking in as large as a twenty-five cent piece. The brain substance was here pulpy, of yellow color, and the limitations or boundaries of the gyrus were effaced. The ventricle was distended by fluid. Ependyma smooth. The area of softening is seen on section to be cone-shaped. It had completely destroyed the left gyrus supramarginalis and extended at its apical portion to the boundary of the ventricle. It consisted of a whitish yellow mass. Aside from this there was found an area of softening about 1 cm. long in the middle of the caudate nucleus, as well a cyst of the size of a pea in the left lenticular nucleus. In the remaining central nervous system nothing striking. There was a general atrophy of the brain associated with diffuse artero-sclerosis.

As it has been often proven that changes more extensive and destructive are found in the caudate and lenticular nucleus without the development of symptoms than those found here, the writer thinks that at first sight we would be led to associate paresis of the vascular system, which was observed in this case with the disease of the supramarginal gyrus, but consideration of the previous physiological data which have been brought to bear on this subject, leads him to the conclusion that the area of softening in the caudate nucleus was the cause of it. In favor of this view are, the fact that the softening in the gyrus supramarginalis was apparently of long standing, the cyst in the lenticular nucleus was sharply circumscribed and seemingly not recent, while the softening in the caudate nucleus was recent.

The writer concludes that the caudate nucleus is a vasomotor centre for the contralateral half of the body.

J. C.

**Two Cases of Raynaud's Disease.**—Urbhart (*Edinburgh Medical Journal*, March, 1895, p. 806).

The author describes two cases of Raynaud's disease occurring in the insane. An interesting feature in both cases is the development of hæmatoma auris and Raynaud's disease in one and the same patient, though at different times. The pathology of hæmatoma auris is still unsettled; its coincidence with Raynaud's disease in these two cases may help to throw light upon the subject.

The post-mortem examination in the first of the two cases showed the absence of any changes in the peripheral nerves and arteries; the veins and capillaries in the area affected were greatly engorged, some of them forming large eminences. There was not much diapedesis, the appearance of hæmorrhage to the naked eye being brought about by the great engorgement of the vessels everywhere with blood. No changes were found in the cord or medulla.

STIEGLITZ.



## Society Reports.

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### THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, held at the New York Academy of Medicine,  
on May 7, 1895.*

Dr. C. A. HERTER, Vice- President, in the chair.

Dr. J. ARTHUR BOOTH presented a case of monocular blindness without any discoverable lesion in the eye. The special point to which he directed attention was, whether after a functional blindness had lasted for a year or two, the morbid condition might not persist, finally causing congestion and inflammation? The patient, Agnes L., fourteen years of age, had been perfectly well up to about one year ago, at which time she had fallen on the ice and struck her head. Two months after this, she began to have headache, chiefly frontal. On May 5, she had come to the Manhattan Eye and Ear Hospital because of dimness of vision. Examination had revealed almost total loss of vision in the right eye, without any change in the vessels or disc. While in the hospital she had had two hysterio epileptoid attacks. Strychnine had been given freely, but without benefit. Vision in the left eye was  $\frac{1}{50}$ ths, but could be brought up to  $\frac{3}{80}$ ths by glasses. The field was much contracted in all directions. In a moderate light, both pupils were dilated, the right being a little larger. There was a marked loss of sensation in the right cornea, but no anæsthesia of the face, body or extremities. Colors were readily recognized by the left eye. The knee-jerks were equal, but somewhat exaggerated. The treatment by means of drugs had failed, as had also hypnotism. Various tests had been made to detect any deception on the part of the patient, but the results had been negative.

## AN UNUSUAL CASE OF LEAD POISONING.

Dr. WILLIAM M. LESZYNSKY presented a man whom he had first seen about ten days ago. He had experienced difficulty in making the diagnosis at first on account of the obscurity of the etiology. The man stated that at the onset, sixteen days before, he had had fever, vomiting and malaise, but there had been no chill, and no bladder or rectal symptoms. He complained of general weakness in the lower extremities. About the same time he first noticed an aching pain in the right shoulder, and two days later, the index and the medius dropped, and the pain disappeared. A few days after this, the weakness had increased, and when first seen by the speaker, there was wrist-drop. The left arm and hand were also becoming weak, but there was no pain. Both extremities had felt numb. There was no history of exposure to cold. For five or six years he had been drinking five pints or more of beer daily. Examination showed muscular weakness—a paresis of the deltoid and upper portion of the trapezius, and the biceps group, and the extensors were paralyzed—as was also the extensor minimi digiti. There was also extreme tenderness along the course of the nerve trunk, as well as some tenderness of the muscles themselves. With such a history one would suspect lead, and there was a slight line upon the gums suspiciously like a lead line, but he was a cooper by occupation, and at first no history could be obtained of his having come in contact with lead. A provisional diagnosis had been made of alcoholic multiple neuritis, beginning in the upper extremities. The urine was then examined, and traces of lead found in it. There was no albumen or sugar. Inquiry then elicited the fact that he had occasion to close barrels containing a preparation of white lead. Electrical examination showed absolute loss of faradic irritability in the musculo-spiral on the right side, and in the extensors, and diminished faradic excitability in the other muscles that were paretic. Both knee jerks were active. Ophthalmoscopic examination was negative. This patient had also tachycardia, the pulse rate being from 116 to 120, and weak.

Dr. M. ALLEN STARR said he had recently seen a man suffering from lead palsy who had been in the habit of drinking a large quantity of water from the faucet every morning. It was believed that the water having

remained in the pipe all night had become impregnated with lead, and that this had given rise to the lead poisoning. The speaker said he had only seen one case of lead poisoning in which all the extremities had been affected nearly equally, and where there had been intense pain along the nerve trunks and muscles. This patient, a painter, had been in the hospital for fourteen months, and all this time had had the typical blue line on his gums.

The CHAIRMAN agreed in the diagnosis of lead paralysis in the case reported by Dr. Leszynsky, notwithstanding the unusual tenderness of the nerve trunks and muscles, but he was inclined to think that the fact of the man having been a steady drinker might have had something to do with precipitating these symptoms. Alcoholism certainly predisposed to attacks of lead poisoning. During the past winter, he had seen a large number of cases of severe alcoholic neuritis, and had been impressed with the fact that in a certain proportion of these the first symptoms had been pain in the joints and in the muscles about the shoulder.

#### AN INTRACRANIAL GUMMA.

Dr. C. E. NAMMACK presented a woman, twenty-four years of age, whom he had first seen two weeks ago. For the past five months she had had constant headache, with vertigo and vomiting in the early morning. This was followed by gradual loss of power in the right hand, followed by motor paralysis of left leg. Two months later she found that she was unable to close the left eye. There had been no injury or emotional shock, and there was no history of alcoholism or of venereal disease of any kind. She answered questions slowly, and it was difficult to keep her attention concentrated on any one subject. There was optic neuritis on both sides, with beginning choked disc in the left eye. *R.V.* was  $\frac{3}{4}$ ths and *L.V.*  $\frac{2}{3}$ ths, not improved by glasses. The tongue was furred on the left side only, and taste was diminished on this side. The diagnosis was an intracranial gumma, localized in the lower part of the pons on the left side. For the past two weeks she had received inunctions of blue ointment, and sixty grains of iodide of potassium, three times a day, and already the improvement was remarkable.

Dr. B. SACHS said that it was well to be cautious

about making a diagnosis of gumma in a case of this sort, solely on the strength of the improvement observed from such treatment. For example, he had seen a man of forty-five years, who had had a hemiplegic attack some years before, and in whom the administration of a large quantity of iodide of potassium had caused the entire disappearance of all the symptoms for several months. Subsequently, this man had a fatal apoplectic seizure, and the post-mortem had shown a very large glioma. In connection with every neoplasm there was a large amount of exudation, and absorption of this fluid might be responsible for the improvement following the treatment.

Dr. STARR said that if he remembered correctly, Dr. Nammack had himself recently reported to the society a similar case. Within ten days he had seen a boy of sixteen, suffering from all the symptoms of well-marked tumor of the cerebellum. When this boy was eight years of age, he had suddenly developed symptoms of tumor of the cerebellum, and had been treated for such a tumor by Dr. Seguin. Within two months after beginning the anti-syphilitic treatment, the symptoms had entirely subsided, and the boy had been apparently perfectly well up to six weeks ago, when general symptoms of tumor and eye symptoms had returned. Examination had shown the typical choked disc. Although this boy had not had syphilis, and the family history was exceptionally clear on this point, yet anti-syphilitic treatment had caused the disappearance of the symptoms of tumor.

Dr. J. F. TERRIBERRY remarked that one could get no idea of the degree of choked disc from the impairment of vision.

#### REPORT OF A CASE OF LEAD PARALYSIS WITH SPECIAL REFERENCE TO CYTOLOGICAL CHANGES IN THE NERVOUS SYSTEM, AND THE DISTRIBUTION OF THE LEAD.

Dr. C. A. HERTER read a paper with this title. The case forming the subject of this paper was Thomas E., twenty-six years of age, poorly nourished and undersized, who had been admitted to the City Hospital on November 23, 1894, and had died on December 12, 1894. His family history was negative. He had used alcohol moderately, but had never had syphilis. He had been a

painter for the last thirty years. In February, 1894, he had been treated in this hospital for very severe lead colic associated with double wrist-drop. With the exception of the latter, the symptoms had entirely disappeared during his stay in the hospital, and he had returned to his work. Shortly before his last admission, he began to have attacks of nausea and abdominal cramps. On admission, he complained of weakness in his arms and legs. Examination showed slight oedema of the lower extremities; apex beat diffused and rather weak; arteries distinctly atheromatous; pulse 80, weak, but regular; well marked blue line on upper and lower gums; well developed wrist-drop; complete loss of power in the extensors of the wrist on both sides, and but little power in the flexors. In the biceps of either side the power was unimpaired. There was atrophy of all the muscles below the elbow, and especially of the interossei. There was no triceps jerk. In the lower extremities, there were slight drop-foot and moderate contracture of the calf muscles. The loss of power was rather more marked on the right side. The thigh muscles reacted fairly well to faradism. The knee-jerks were feeble on both sides. No ophthalmoscopic examination was made, but the acuity of central vision was distinctly reduced. The patient was kept in bed, and received, by mistake, ten grains instead of five grains of iodide of potassium, three times a day. Two days afterward, the daily quantity of urine was 1,600 cc., it was acid, had a specific gravity of 1.016, and contained half a gramme of albumen to the litre. On December 6, there were 670 cc. of urine excreted, and it contained half a gramme of albumen to the litre: urea 14.36 grm.; uric acid .17 grm.; phosphoric acid, 4.86 grm.; sodium chloride, 1.7 grm.; pre formed sulphates, .80 grm.; combined sulphates, .20 grm. The absolutely low urea, and relatively and absolutely low uric acid were striking features. On December 7, the patient began to be drowsy during the day, and three days later he was in a muttering delirium, and the pupils were contracted and reacted but little to light. On December 10, the pupils were pin-point, the respiration was stertorous, the eyeballs prominent, the pulse feeble, and the body surface cold and clammy. Death occurred on December 12. On the last day, 410 cc. of urine were drawn by catheter. It had a specific gravity of 1.015, and contained albumen and a considerable number of granular and epithelial casts.

The ratio of uric acid and urea was 82.7; the chlorides were low, and the sulphate ratio was again exceedingly low. Blood, drawn two days before death, showed the percentage of urea to be .76, which is two to three times the normal amount. At no time were there convulsive movements.

Dr. IKA VAN GIESON described the results of the post-mortem examination. He said that attention had been paid chiefly to the cytology of the ganglion cells—in other words, to the science of these cells. It was only by such investigations that we could fully understand the changes underlying a great many nervous disorders, and particularly the toxæmias, and affections due to over-fatigue of the ganglion cells. By reference to a chart, he showed how complex was the structure of these ganglion cells, as determined by the modern methods of research. Lead poisoning, he said, should be one of the most brilliant fields for this new line of observation, as the process was so slow. At the autopsy on the case under consideration, several erosions were found in the brain. The vessels at the base were normal, as were also the cranial nerves. The viscera, with the exception of the kidneys were practically normal. The surface of the kidneys was rough and granular, the cortex was very thin, and the topography of the tubules was lost. The submaxillary glands, the thyroid and the suprarenal capsule showed no significant changes. Dr. Herter had found the epithelium of the secreting tubes in a state of marked granular degeneration, that the cells of Henle's loops were degenerated, and contained hyaline casts, and that the cells of the collecting tubes were degenerated and proliferated, and their lumen filled with degenerated cells and hyaline material. About half of the glomeruli were in a state of advanced fibrous atrophy; in the remaining glomeruli, the capsule was thickened, the capillaries compressed, and the cells of the tufts moderately increased. The tufts filled up the capsules.

The peripheral nerves had been hardened in osmic acid, and examined with the following result: In the ulnar, about one fibre in every ten was moderately degenerated; in the external peroneal, about one-fourth of the fibres were collapsed and degenerated; in the sciatic of the middle of the thigh, about one degenerated fibre was found in every ten; in the left external cutaneous, there was partial degeneration of one fibre in ten, and more complete degeneration in three fibres in ten; in

the right radial at the wrist, one fibre in twenty was degenerated; in the left plantar, one in twelve.

Portions of both enlargements of the cord were hardened in various ways, and examined in detail. By applying anilin dyes, fully one-third of the ganglion cells showed a form of degeneration which changed the internal composition of the cell, although not its shape. Comparatively few of the cells were completely destroyed. The changes observed were not apparent if specimens had been hardened in Müller's fluid, and stained with carmine. In the specimens examined, the chromophyllic granules had been broken up and disseminated throughout the ganglion cells. Such changes he thought could be considered as entirely independent of the changes observed in connection with rest and fatigue. Some of the cells showed beginning vasculatation, and others an accumulation of pigment. It was now known that the quantity of pigment indicated the age of the cell. Where the degenerative process had gone still further, the cells had been nearly destroyed. These cells had been found equally distributed in the cervical and lumbar enlargements. Fully one-third of the ganglion cells showed the mild changes first described as occurring in them.

In the sympathetic system no such changes had been as yet detected. The cerebellum in this case had been found absolutely normal. The motor zone had been particularly examined in the hand region, but as little work had been done on the normal ganglion cells here, no positive deductions could be made. It was to be remembered that in cases of toxæmia there was often a rise of temperature shortly before death, and this might produce some of the changes observed in the nervous system.

Dr. HERTER, continuing, said that the various organs or portions of them, had been oxidized as thoroughly as possible, and the lead separated from this material on platinum plates. From these plates it had been dissolved with nitric acid, and ultimately converted into a lead chromate, and weighed as such. The spleen contained .004 per cent. of lead; the kidney, .0004 per cent.; the liver, .0021 per cent.; the brain, .001 per cent.; the spinal cord, .0031 per cent. The lymph nodes of the mesentery gave no lead reactions. The soleus muscles contained .0081 per cent.; the lungs, .001 per cent.; the heart a trace, and the blood, contrary to the experience

of other writers, contained .0002 per cent. of lead. The brain, spinal cord and liver also contained small amounts of copper.

The speaker said that the case was of interest on account of the loss of power in the upper and lower extremities, and the long time the patient had been exposed to the influence of lead without exhibiting symptoms of lead poisoning, and also because of the cerebral symptoms shortly before death. The great preponderance of the lesions in the spinal cord over the peripheral nerves was, perhaps, the most striking feature of the post-mortem findings. It was to these spinal lesions that the atrophic paralyses must be referred. The changes in the lumbar enlargement were rather more marked than in the cervical enlargement.

Were the cerebral symptoms to be considered as due to the direct action of the lead on the brain? Three points should here be considered, viz.: (1) The motor areas showed no distinct change in structure; (2) the motor areas contained a small amount of lead, and (3) the fact that there were only slight changes in the cortex, which did not exclude the possibility that the cerebral symptoms were due to lead. Two cases had been reported going to show that the cerebral symptoms might be present, even without there being any lead in the brain. A number of facts gave support to the theory that these cerebral symptoms were uræmic in character. The kidneys in the case reported must have been taxed to their full functional capacity for a considerable time before death. The post-mortem findings indicated that an acute diffuse nephritis had been engrafted on a chronic nephritis. The clinical record of the condition of the urine would also seem to support this view, for just before death the elimination of urea was defective. Again, the blood drawn just before death showed a marked increase of urea. It might be assumed, therefore, that the retained urea and various leucomaines were concerned in large degree in the production of the cerebral symptoms observed shortly before death.

The evidence strongly suggested that the chronic diffuse nephritis in this case was the result of the lead poisoning. Many observations had certainly shown that this might be a cause of the small, contracted kidney. In this case, there was nothing to indicate that there was any true gouty element present. The acute nephritis was possibly induced by the irritation of the kidneys



resulting from the administrations of the large doses of iodide of potassium and subsequent liberation of lead. The spinal cord contained nearly three times as much lead as the brain. There was no significance to be attached to the small quantities of copper found in the brain.

## DISCUSSION.

Dr. L. STIEGLITZ said that some years ago he had made some extensive experiments on rabbits and guinea pigs with lead. He was gratified that the lesions described by Dr. Van Gieson confirmed his own results. Besides gross lesions in the anterior gray columns in two or three cases he had noted the vacuolation of the ganglion cells in those animals in which the paralysis had been most marked. Erb, in a paper on lead palsy, had claimed that the anatomical changes in the peripheral nerves did not settle the pathology of lead palsy—indeed, he went so far as to say that in lead palsy there might very well be first of all a functional lesion of the ganglion cells. By "functional" he meant changes so minute as not to be detected by the technique of the time; it was gratifying that the refined methods of the present day applied by Dr. Van Gieson have demonstrated these very changes.

Half of the animals he had experimented on had died of cerebral hæmorrhage. The probability that the condition of the kidney was responsible for the cerebral symptoms observed in lead poisoning, had already been urged in a paper by Westphal. It was well known that in acute poliomyelitis of children, a certain part of the paralysis was entirely recovered from, and there was no reason to doubt in these cases that the ganglionic cells of the anterior horn were affected, probably by minute lesions similar in character to those just described by Dr. Van Gieson.

The determination of lead in the different organs was a very crude method, and one should be cautious about deductions found on such determinations; an organ like the spleen, for instance, might contain a great deal of lead without functional disturbance, and the organ more sensitive to the poison, like the cord, contain very much less and still suffer to a greater degree.

He believed the pathology of lead palsy could only be decided when we had found a microchemical bath for lead, for we could then demonstrate the lead in the

degenerated ganglionic cells or nerve fibres, as the case might be.

He had devoted considerable time to observations on the kidneys of animals and had found both chronic and acute nephritis.

Dr. MARY PUTNAM-JACOBI thought that the facts brought out to-night showed that the lead acted, not after being deposited in an organ, but during its circulation through the organ and by interfering with the metabolism of its tissues. Thus the nutrition of the ganglion cells might be seriously depressed, and yet only a minute quantity of lead be detected in nerve tissue. The frequent association of gout with lead poisoning was another indication that the lead acted by disturbing metabolism. Nephritis was probably induced by the irritation of organic poisons thus formed, rather than by direct action of the lead on the kidneys. This view would seem to be more in consonance with our knowledge of the comparatively indifferent nature of lead.

Dr. HERTER, in closing the discussion, said, he agreed with Dr. Jacobi that the small, granular kidney was produced in cases of lead poisoning by an indirect influence on the metabolism of the body, resulting in an increased amount of work being thrown on the kidney. He had not been aware that Westphal had stated his belief that lead encephalopathy was due to the condition of the kidneys, although he knew that Oliver had accepted this view for a certain small number of cases. He did not think, however, that anyone previously had demonstrated that the blood in these cases did actually contain a marked excess of urea and allied substances.

## Book Reviews.

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RELATIONS OF DISEASES OF THE EYE TO GENERAL DISEASES. By Max Knies. Edited by Henry D. Noyes. Wm. Wood & Co., Publishers, New York.

This book, which has for some time been well known to students of neurology and optahalmology and appreciated by them because of its being a veritable store house of information, is now made accessible to the English reading practitioner. Although, as the title indicates, the purpose of the book is to discuss the relations of diseases of the eye to general diseases, more than one-half of the entire volume is taken up in discussing the ocular relations and manifestations of nervous disease. The importance of recognizing the information to be derived from a careful examination and study of the eyes is patent to every neurologist, and the fact that it is necessary to give the subject such a large amount of space in a comprehensive work of this kind must bring the truth of this statement home to the general practitioner.

The author describes in some considerable detail the anatomical course of the nerves of the eye, the optic, the motor and the sensory, and the disorders in the domain of these nerves and their central origin. His statements on these points are a commendably accurate reflection from the literature, without, however, having being subjected to a very critical sifting. In commenting on the region of primary and direct cortical visual impressions, the editor very pertinently remarks that later investigations have shown that this region is confined to the calcarine fissure which is the inferior boundary of the cuneus. We miss a reference to what may be called a new conception of the termination of the optic nerve in the retina, one of the advances which we owe to the Golgi and Cajal stain, and which does away with the conception of the many layered retina. We miss also a discussion of the connection between the lower optic centres and the grey matter of the bulb and cord, a knowledge of which is sometimes absolutely necessary in order to explain frequent anomalous ocular symptoms in the course of a nervous disease. Also a mention of the connection between some of the fibres of the optic tract and the cerebellum, a fact which has been brought out by the Marchi method of investigation, and which must be invoked to explain some of the eye symptoms of cerebellar disease. As a whole, however, these chapters are very satisfactory and may be taken as a trustworthy statement.

The pages devoted to disorders of the voluntary ocular muscles are among the most lucid in the book, and the space given to the discussion of nystagmus is well deserved. The causes of nystagmus may be peripheral, central, or both, and the condition itself may be defined as imperfect cortical innervation of the voluntary muscles of the eye; but we believe it to be unfortunate that the author finds it necessary to say that the condition is a peculiar form of paralysis agitans, as such a designation adds nothing to the lucidity of the matter, and it may cause confusion. Some of the wood cuts used to illustrate the anatomical basis of trans-cortical disorders of vision are not by any means what they should be, and one or two of them come very near to being entirely misleading. For instance, an illustration on page ninety two shows the first frontal convolution adjacent below to the fissure of Sylvius; that is, in a loca-

tion where the third and fourth frontal convolutions are. The second frontal is depicted as occupying the position which should be given to the first, while that which is rightfully called the second is put down as the third.

In fact, it may be justly said that many of the illustrations are lacking in accuracy.

The author says that inasmuch as he regards the frontal part of the brain as superior to the remainder of the cerebral cortex, he is opposed to the majority of writers who consider such a higher centre unnecessary and superfluous. Just what he means by this is not easy to say, for most persons would willingly admit that he who contended that the frontal brain was unnecessary and superfluous was very lacking of that part of the cerebrum. If it is meant, however, that this part of the brain is necessary for the performance of the ordinary functions, we can see that he is opposed to all physiologists.

The eye in relation to the individual diseases of the brain, cord and nerves is discussed very specifically and with a great deal of care; but here, as in some other parts of the book, we concede an impartial presentation of facts, or fiction, as it may be, but we miss the personal impress of the author. For instance, in speaking of the ocular conditions in anæmia and hyperæmia of the brain, he says, apart from the ophthalmoscopic findings, the symptoms of cerebral congestion are usually said to be restlessness, irritability, diffuse, headache and narrow pupils; those of cerebral anæmia are drowsiness by day, sleeplessness at night, circumscribed headache and dilated sluggish pupils. That is, unfortunately, what some one wrote many years ago and which has fastened into literature like a barnacle on the bottom of a boat; but we are evolving out of it, as it were. The reviewer has never seen a patient on whom a diagnosis of cerebral anæmia or hyperæmia has been made, and some day it is probable there will come an author who will write a book without describing this condition, and thus gradually shall we become emancipated from this bit of imagery.

In speaking of cerebral hæmorrhage, the author says that very often there is also homonymous hemianopsia in the visual field of the side opposite to the hæmorrhage, but this usually disappears in a short time unless the visual zone or the optic radiations in the corona radiata are the site of the hæmorrhage. The student reading such a passage will be puzzled to know just what area is indicated by the words "visual zone."

The pages devoted to abscess, tumors, meningitis are an excellent expression of the consensus of opinion held by ophthalmologists and neurologists concerning the form and frequency of eye symptoms in these conditions. The eye symptoms of tabes are given liberal space and discussion; but in this chapter particularly, obscure phraseology, lack of continuity in expression and involved sentences often obscure the meaning of the writer. For instance, he says "muscular disorder of all kinds occur with equal frequency in tabes." It is to be inferred that eye muscles are meant. Again he says, "pronounced inflammatory or hæmorrhagic inflammatory phenomena; for example, inflammation of the ependyma of the fourth ventricle and hæmorrhages into the adjacent gray matter do not belong to tabes proper, but to multiple neuritis." This will strike neurologists as being rather novel. Again, on page 196 he says, "The bulbar and cerebral nerves were also attacked finally in a case of anterior chronic poliomyelitis." Other expressions, such as ocular facial, choked neuritis, cerebral nerves, etc., are frequently met with. The consideration of the ocular symptoms of that group of phenomena classed under the traumatic neurosis are the most unsatisfactory in the book. The author evidently has not the recent conception of these conditions as railway spine and the traumatic neurosis are considered under entirely separate captions.

If we were asked to give the most palpable shortcomings of the book, we would say the absence of the critical faculty which the author manifests in considering and summing up the various statements or claims that have been put forth by as many different writers. It would be a much more lengthy statement to give the book's principal claims for the recognition and careful attention of the general practitioner and specialist, for they are many. The volume will prove undoubtedly to be a satisfactory and reliable source of reference for those who would get the opinion of many.

The volume gives evidence of having been carefully edited, and is well printed, but the translation is not all that it might be. J. C.

DEGENERATION. By Max Nordau.<sup>2</sup> D. Appleton & Co. New York, Publishers.

Each succeeding year brings with it a book which seems to engross the attention of the talking public, at least for a short time, to the exclusion of everything else. Last year it was Kidd's *Social Evolution*, this year it would seem to be Nordau's *Degeneration*. We are probably not mistaken in considering it one of the most widely-read books of the season. It deals with a subject which has a personal interest to every one and to everyone's neighbor. It has received consideration from distinguished critics and thereby has had distinction given to it. It has called forth opprobrium from some who have fallen within the lines of its strictures and in a general way it has succeeded in making itself felt, whether to any purpose or not, beneficial or detrimental, cannot be said with safety.

The author takes himself most seriously, both in his dedication, which is rather fulsomely made over to Lombroso, whom Nordau respectfully calls master, and in his text.

The volume is comprised in five books, the first being a disquisition on *Fin de Siècle* considered generically and specifically. From the very first line the author shows a vast familiarity with contemporary fact and fiction and draws largely on his knowledge and on his note-books. From the start he manifests a predilection to rap the country under whose flag he was bred and palliate the land of his domicile. Another fact which must strike the most casual reader the minute he has turned the first few pages, is the truly phenomenal power of vituperative, and the wealth of expletive and explosive which the author is possessed of. This conviction will grow on the reader as he turns the pages, as will also the feeling that the centre in the author's brain in which this element of his mentality has its seat is very easily inhibited, for the slightest provocation, or no provocation at all, calls forth a torrent of oburgation and contumely which makes one gasp. For instance, after speaking of the "change of fashion in the arrangement of the coiffure, a somnolent subject at its best, he says, "The majority anxious to be inconspicuous in unimaginative mediocrity, seems to have for its leading style a labored rococo, with bewildering oblique lines, incomprehensible swellings, puffings, expansions and contractions, folds with irrational beginning and aimless ending, in which all outlines of the human figure are lost and which causes women's bodies to resemble now a beast of the Apocalypse, now a Aripitych, etc.

For the benefit of our readers we may say that the author of *Degeneration*. Dr. Max Simon Nordau, was born in Buda-Pesth, Austria, July 26, 1841. He was educated for a physician, received the degree of M.D. in 1873, spent several years in travel in Europe, went to Paris to visit the Universal Exposition of 1875, and continued his medical studies and made his permanent home in that city. He became a correspondent of the *Revue Française*, the *Frankfurter Zeitung*, and the *Leipziger Zeitung*, wrote several feuilletons and other articles for various French journals and reviews, and began publishing critical, political and social works, many of which attracted wide attention.

Again, the general reader cannot proceed to any great length before a feeling of surprise comes to him that it is scarcely credible that the sober English language should be made the vehicle for some of the sentiments which are here put down, for instance, "Elegant Titillation only begins where normal sexual relations leave off." Nothing seems too mean or loathsome to be dished up in plain, homely Anglo-Saxon. A poor, irresponsible and insane king of Bavaria must needs have it told of him that he was wont to have religious concerts given in darkened rooms so that religious music might augment sensations from hidden enjoyment of another sort.

In the *Fin de Siecle* there is not, apparently, the faintest ray of anything commendable, especially of the origin, dress, habitation, animation of the upper element. Degeneration and hysteria are their "possessions." We have no desire to contend this with the author, but for the sake of our overworked friends we would demur to neurasthenia being called a minor stage of degeneracy or a forerunner of hysteria, for neurasthenia is no more a minor stage of hysteria than rheumatism is a minor stage of gout.

In fact, in discussing the purely technical side of the question of mental disease and degeneration, it cannot be conceded that Mr. Nordau is at his best. For instance, he says the two psychological roots of moral insanity are, firstly, unbounded egoism, and secondly, impulsiveness. If impulsiveness is used in the sense of obsession, yes; but if it is used in its colloquial sense, emphatically no. Candor compels us to say that Nordau has no superior in seeing one side of a question, and he is easily first if it happens he wants to see that side. It is really refreshing to be told that it is a prominent symptom of degeneracy for one who has no claim to the title to style himself "artist," for we have long had doubts of some German barbers and Italian bootblacks who are thus styled, self-styled.

Although we are not moved to any serious inclination to join forces against Mr. Nordau in his symptomatology of degeneracy, it does not seem to us that he can substantiate his statement that "from a moral point of view their (the degenerates) existence is completely deranged. If anything has been whispered against the moral lives of Ibsen and Zola who in Nordau's eyes are mirrors held up to degeneracy, the whispering has come to the ears of the few and not the many. And again, if it were necessary, it would, we think, not be an extremely difficult task to refute many of the statements concerning the evidences of degeneracy to be found in the dramas of Ibsen, the poetry of Rosetti, the music of Wagner, and much of the art of the Decadentists; themes which furnish Nordau opportunity for revilement. It does not behoove us to follow out such an inclination in this place, yet we cannot stay our hand from transcribing the closing passage of a critical essay on Dante Gabriel Rosetti from the pen of a critic whose like as a master of style and authority in literature has not been excelled by any man of his years during the present generation, Walter Pater: "Rosetti did something, something excellent . . . ; but his characteristic, his really revealing work, lay in the adding to poetry of fresh poetic material, of a new order of phenomena, in the creation of a new ideal."

It is impossible to follow the author through his large book of upward of 500 pages, scarcely a page of which might not be commented on, either adversely or favorably, nor would it be just to our readers, who will get some edification, not a little enjoyment and a modicum of disgust from a personal examination of the book.

The feeling which we have personally about the matter is, that it is really a misfortune that the author could not have brought to the prosecution of his work a really scientific and critical spirit and allowed these to temper and guide his apparent erudition and wide reading. He has demonstrated a lack of these faculties and the misfortune is the greater

that he did not have the perspicacity to submit his ebullition to the cooling influence of a scientific co worker. In such a case we believe the result would have been a different one and one productive of beneficent and lasting results. As it is now it would seem to be corroborative of that bit of gamin philosophy which is contained in the question and answer, "What's the good of anything? Nothing."

In concluding these remarks which we make merely to direct attention to the book, we are reminded by way of contrast of some remarks made by Sir Edwin Arnold anent two American degenerates—Nordau a few years ago in an after-dinner speech at one of the clubs in this city. That speech which seemed to its auditors remarkable alike for its fairness and erudition contained an allusion to "The catullus-like perfection of the lyrics of Edgar Allen Poe, and the glorious large-tempered dithyrambs of Walt Whitman." To Sir Edwin the former was more than jingle-headed and the latter not the blatant idiot of Nordau.

But then we remark what can be expected from one who has the bad taste, to call it by no worse name to stigmatize a contemporary, who at least has some admirers, as an incurable cretin? JOSEPH COLLINS.

**NERVOUS DISEASES OF CHILDREN.** By B. Sachs, M.D., Professor of Mental and Nervous Diseases in the New York Polyclinic, etc. 647 pages, with an appendix. 162 illustrations. Wm. Wood & Co., Publishers, 1895.

This is the first attempt that we are aware of, in this country at least, to scientifically classify and study the various nervous disorders which begin in infancy. The author's long experience and close observation particularly fit him for the task he has undertaken and the result of his labor is unquestionably a valuable addition to American neurological literature.

The study of nervous diseases in children is for many reasons vastly more difficult than in the adult, and, therefore, a more refined and intimate knowledge of the various methods of detecting abnormal conditions is necessary than the average physician is at all likely to possess. For this reason the introduction, which treats of the various methods of examination, is one of the most important and valuable divisions of the book. Simple yet reliable tests are described for detecting abnormalities of sensibility; directions for ascertaining disorders of the special senses are concisely explained and the relation of paresis and paralysis to disease of the cerebro-spinal system have been carefully elaborated in a series of tables which deserve more than a passing notice. These tables contain in parallel columns the names of the various important muscles of the body; their normal functions, the symptoms exhibited when their actions are deficient, the name of the nerves which supply them, and the locality of their representation in the cord or medulla, and finally the forms of disease which commonly depend upon the preceding conditions. In many instances, and especially where the diagnosis is at all doubtful, these tables will be found invaluable.

The chapter ends with a few words relative to the employment of electrical currents for diagnostic purposes. The author points out that the main utility of electricity consists in differentiating between certain diseases involving the anterior horn cells and the cranial nerve nuclei, and their projections on the one hand, and diseases of the rest of the cerebro-spinal system on the other. Tables explanatory of the reactions of degeneration and of the diseases in which these degenerations are found, close a most instructive and entertaining chapter.

The first part of the treatise on the diseases of the nervous system begins with the general nervous diseases and the so-called functional neuroses. This section treats of such affections as convulsions, hysteria, chorea and allied mobile spasms, tetanus and tetany, headaches, the disorders of sleep, and the vasomotor and tropho-neuroses. Whether there are, strictly speaking any functional neuroses is a question which will possibly, sometime in the future, be answered negatively. Diseases which are curable and in which no structural changes have been discovered in the central nervous system are usually described as functional neuroses. This term has been retained by the author, and very properly so, for although the most recent methods of investigation disclose structural changes in the nervous system which could not previously have been detected, the time has not yet come, though it probably is coming, when the morbid changes producing the so-called functional neuroses can be definitely ascertained. This section contains a clear and concise history of the numerous affecti us discussed in it; the pathology and pathological anatomy is ably set forth and the treatment advocated is that which has proved most serviceable in the author's experience.

The second part of the work is devoted to the consideration of organic diseases of the nervous system. Diseases of the peripheral nerves, including brachial plexus lessions, obstetrical palsies, facial paralysis, tic convulsif and wry neck, and multiple neuritis, which takes up the entire succeeding chapter, are treated with the author's characteristic thoroughness.

Diseases of the spinal cord are introduced by a chapter on the anatomy, physiology and pathology of that structure. When volumes as large as the one under consideration have been written on the anatomy of the spinal cord alone it is impossible for a writer of a text book on general diseases to do more than refer briefly to the most important facts. This the author has accomplished most successfully. The various diseases of the spinal cord are satisfactorily discussed. The chapters on Infantile Spinal Paralysis, Syphilis of the Spinal Cord, and Hereditary or Family Diseases of the Cord are particularly worthy, especially the latter chapter, which treats of Friedreich's disease and the hereditary forms of Cerebellar Ataxy and Spastic Paralysis. The succeeding chapter on Progressive Muscular Atrophies is also worthy of mention. The differentiation of atrophies of central origin from primary myopathies, both from a clinical and histological point of view, is exceedingly well expressed.

The chapter on the anatomy, physiology and pathology of the brain, though but a short review of well-established facts, is carefully compiled and demonstrates the most important features of these subjects.

The author has evidently taken great pains in the preparation of the several chapters on diseases of the brain. Those on "Infantile Cerebral Palsies" and "Diseases and Conditions due to Defective Development of the Brain" are particularly exhaustive and instructive. Both of these subjects have engaged the author's attention for several years, and the elaboration of his views in the present volume will, therefore, command more than ordinary attention.

The work closes with a chapter on insanity, one on idiocy and imbecility and an appendix containing a few therapeutic suggestions. Of course, it is impossible to treat the subject of insanity, even the insanity of infancy, with anything like thoroughness in the few pages that can be spared for the subject in a work like the one under consideration. Nor does the author attempt more than to give a mere sketch of the different forms of insanity and to suggest the most appropriate methods of treatment. The chapter on idiocy and imbecility is very thorough and complete.

Taking the work as a whole, there is much to commend and but little to condemn. It will promptly take its place in the foremost ranks of neurological literature.

GRENE M. HAMMOND.



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and 7, 1895.*

DR. PHILIP COOMBS KNAPP, of Boston, President, in  
the chair.

*Morning Session at 10 o'clock.*

ADDRESS BY THE PRESIDENT.

PHILIP COOMBS KNAPP, A.M., M.D.,

Of Boston.

GENTLEMEN:—Our programme is long and contains much that is more interesting than anything I can offer. One or two duties, however, must be performed before I can invite you to listen to it. First of all, I thank you for the honor conferred upon me in choosing me to preside on this occasion. Next, I must offer to you, in the name of the medical profession of Boston, a most hearty welcome on this your first visit as an Association to our city, with a wish that you may pay us many more such visits; and finally, I must present to you greetings and regrets from our honorary and associate members abroad, Erb, Hughlings Jackson, Ferrier, Horsley, and others, whose letters will be read later. Having done this it might be more fitting to begin upon our programme without delay, but, in view of the fact that the Association has just completed its twentieth year of life, I may perhaps be pardoned for detaining you for

a few words of retrospection and of suggestion for the future.

On the fifteenth of December, 1874, a letter was sent out, signed by seven men, three of whom are still active members of this society. Its opening sentence was as follows: "It is contemplated to institute a society, to be called The American Neurological Association, to be devoted, as its name imports, to the cultivation of neurological science, in its normal and pathological relations." Five and thirty men, including the signers, accepted the invitation, and on the 2d, 3d and 4th of June, 1875, the first meeting of the new Association was held in New York. Twenty members were present, and Dr. Weir Mitchell was elected as the first president, but, on his withdrawal, the late Dr. Jewell was chosen to fill his place. The new society was something of an experiment; there were but two similar associations in this country the Ophthalmological and the Otological, where there are to-day thirteen or more. Few neurological societies existed abroad, and it was not until eleven years later that the Neurological Society of London was organized. Up to that time, in most parts of the world, the neurologist had had to content himself with the crumbs that fell from the alienist's table, and those, apparently, had been so scanty that at first we declined to share our own feasts with the alienists. We have grown more liberal with years, so that we have even offered them of our abundance of late, although the food was not wholly to their relishing. With that exception, which was but temporary, we have pursued no exclusive policy. Although dealing with the highest and most difficult problem of medicine, the study of the brain of man, ours is not the speciality of a single organ; we must search every part of the body to make our diagnosis, and be general physicians first and neurologists afterwards. We went out with our first invitation into ophthalmological highways and hedges, and we have ever welcomed the physiologist, the general physician, and the surgeon into our ranks.

I must leave it for others to speak of those early and, if tradition be correct, somewhat stormy days of the Association's life. A number of the founders of the society are happily with us, who have taken honorable part in the transactions during the whole twenty years; we regret the absence of the rest who are still on our rolls, — Bartholow, Bannister, Mitchell and Seguin. Others, too,

can speak from better knowledge of those who met twenty years ago and who have now finished their work—Jewell, Hun, Clarke, McBride and Van Bibber. A word of tribute, however, is due to one of our later members who has died this year, that genial comrade Matthew Dudley Field, who sent, only a week before his death, a cheering message, full of hope that with restored health he might be here to day.

Of the work which we have done during the last twenty years little need be said. We have flourished as a society, our membership has increased from thirty-five to eighty-five, our meetings have steadily grown in interest and in profit, and we have done our share in the establishment and the success of the most important medical body in this country, the Congress of Physicians and Surgeons. Of the scientific work which has been done under the auspices of the Association it can honestly be said that if, both in neurology and psychiatry, the work done by our members in the last twenty years were to be deducted, America's claim to consideration in the scientific world in those branches would be poor indeed. We have done something to add to the enormous mass of knowledge concerning the nervous system that has been accumulated during the last twenty years. What the advance in that knowledge has been we all know, and time is lacking to review it here.

Although on such an occasion as this a little self-gratulation is pardonable, it is the part of wise men not to indulge in the vain-glorious boastings of the Fourth of July orator, but to ask ourselves not what we have done, but rather what we have left undone. As we look over not only the more complete bibliographies, but also the various references in the works of our colleagues abroad,—where, by the way, we find much more frequent notice of American work than there was twenty years ago,—we may observe that American names are far less frequently quoted in the sections on anatomy and experimental physiology than in those of symptomatology, pathology and therapeutics. Our programme to day shows that we are doing some work in these directions, but the amount that has been done is still below the mark. We have as yet produced no work on anatomy to set beside even the elementary work of Edinger, much less the exhaustive treatise of Dejerine,—to cite only one or two examples,—nor have we produced any work on the experimental physiology of the

nervous system to rank with those of Munk, Ferrier, Luciani, or Goltz. In these branches we have done too little, but, in extenuation, it must be said that it is only within a few years that the facilities for experimental research have been at all adequate, even in our larger cities, and we are still in great need of endowments sufficient to carry on our research. The beginnings that we have made show that when it shall become possible for a young man to make a living, though but a plain one, from purely scientific work in the field of neurology, we may expect results which will put us more nearly on an equality with our foreign brethren.

We need make less apology for the clinical and pathological work that we have done in the last twenty years. It has, perhaps, been given to but few of us to attain the grisly immortality which links our names to some new form of disease, but we have added a few more titles to the list of diseases and their symptoms, and we have done much to define more accurately and to describe more in detail the affections discovered by others. As might be expected from the eminently practical nature of the American mind, our greatest additions to the world's knowledge have been made in the domain of therapeutics. The contributions of American neurologists and surgeons have done much to teach us the all too narrow limitations of the operative therapeutics of the nervous system. But for an oversight, we should have been first in the field in the operative treatment of cerebral tumors, and we can claim second place on account of the character and amount of the work that has been done in that field in this country. We have done something, perhaps enough, to prove that neither hypnotism nor treatment by the animal extracts is likely to prove a panacea for the affections of the nervous system. Important, too, as it may be to discover a new stain or define a new tract of fibres in the pons, our chief aim, after all, is to heal the sick, and, in spite of the promise or lack of promise of brain surgery, animal extracts, electricity and hypnotism, there has been no discovery as yet in these twenty years in the domain of the nervous system, that has brought comfort and healing to so many people as that of the rest cure by one of our active, and soon, I hope, to be honorary members, Dr. Weir Mitchell, of Philadelphia.

So much for our past. We need in the future a journal of neurology under our own control, with a sufficient

financial support to enable us to publish fitting illustrations with the reports of our anatomical and pathological work. We need, too, in most of our cities, that recognition in our large hospitals which our president urged last year, a recognition which will come when we have taught the public the necessity and importance of our special field of work. While we are teaching, too, let us do something toward making the general physician realize that the ignorance of neurology, which he too often professes, is far from creditable, and that it would render him ridiculous in any country but this.

The problems before us which demand our attention are too numerous to be rehearsed. Much of the brain cortex still resembles the map of Africa as it used to be in our boyhood—an unexplored blank. We must apply the Golgi methods in disease, and define, if possible, the pathology of the neuron. We must banish the word functional, by demonstrating the pathology of the so-called functional diseases. We must study still more precisely the symptoms of disease, and put our knowledge of electrical diagnosis and the sensory disturbances on a more exact basis. We must differentiate the various clinical forms of insanity, and bring some order out of the chaos labelled neurasthenia. Above all we must remember, as has just been said, that our chief aim is to heal the sick. What shall it profit our patients, if we know the course of the axis cylinder of every neuron in their spinal cords, and the exact percentage which each factor has in the ætiology of tabes or general paresis, or the finest points in differential diagnosis, or the minutest pathological changes, if the end of it all is to be a Weigert stain?

It is too much to hope that in the future we can remove sclerosed tissue and substitute healthy nerve cells or fibres in its place, or that we can do more than alleviate for a time the symptoms which such sclerosis causes. Our hope, therefore, must be in prevention.

The discussion which we held last year at the Congress in Washington indicated the importance of infection in the ætiology of many disorders of the nervous system. In the work of the bacteriologist and the boards of public health lies much hope for the future. When we can deal with syphilis and tuberculosis as we can with small-pox, and as we seem likely to do with diphtheria, we shall have done much toward the prevention of some of the most hopeless affections of the nervous system.

For those affections dependent upon a defective nervous organization, an unstable and invalid brain, much has already been done, and still more can be done in the future when the public is ready to admit the unpleasant fact that such persons are mental cripples, and must have their lives ordered for them as the surgeon now orders life for the hunchback or the cripple. They need a definitely prescribed existence, as to their food and drink, their stimulants and sedatives, their work and play, their study and exercise, their reading and society. This is done now in a few cases, but we see daily many other cases where it is in some degree advisable, and where, if it were done, many morbid nervous and mental conditions might be prevented, or at least materially benefitted. For such people and for many who call themselves well, the conditions of our social life are injurious, but we are none of us young enough to hope to change the entire social fabric. There is very much in the world that does no harm to a well man, which these people cannot bear; we cannot alter the world to suit them, but, when we are permitted, we can shield them from the special things of the world that prove injurious.

In yet another way we can protect the unstable and those who are not sufficiently developed mentally to act absolutely as their own rulers, and thus we can prevent a certain amount of nervous disease. As our boards of health strive to protect the public from infection by specific germs, so it should become our province to guard the public against mental contamination from the degenerates of whom we are just now hearing so much. We restrain the lunatic who has homicidal impulses; we should also strive to counteract the evil influences of the fanatical, neurotic, and degenerate, in the fields of sociology, politics, morals, religion, art and literature, and point out their morbid tendencies.

We have heard something too much for a number of years of the increased nervousness of our age. The neurotic and the degenerate, like Job, speak in the anguish of their spirit, and complain in the bitterness of their souls. They tell us that the world is sick with their disease, and, with damnable iteration, they analyze, not only for their physicians but for the public at large, all the mental, moral, physical, and sexual aberrations of their diseased minds. It is a matter of doubt, however, how much harm has been done to the nervous sys-

tem of the healthy man by the penny post, or the telegraph, or the railway train, or even the daily paper. Let us admit, however, that in the struggle for existence as manifest in our day, the weaker brain must succumb, that social and mental unrest are great, yet, if we look back a little, we find throughout history the same struggle, perhaps under different conditions, the same unrest, the same doubt, the same *tedium vitæ*. The preacher two thousand years ago held that all was vanity and vexation of spirit, yet, even then, he found no new thing under the sun. With the diminution of infectious diseases, better food, more rational standards of living, and greater security for life and property, it is more than probable that there has been an actual diminution in the total amount of nervous disease, even though greater knowledge permits us to detect the less marked manifestations of such disease. Greater or less though the amount may be, there is still much that is distinctly preventable,—let it be our aim in the future to aid in its prevention.

# HYSTERICAL AMBLYOPIA AND AMAUROSIS. REPORT OF FIVE CASES TREATED BY HYPNOTISM.

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## ABSTRACT.

WE may define hysteria to consist of such a condition of the general nervous system, original or acquired, as renders it capable of simulating most local diseases; of complicating them in their progress and modifying them in their usual phenomena. The number of derangements and diseases which hysteria is capable of simulating is well known; but defective vision is one of the less familiar forms, and so I take the opportunity to direct your attention to this subject; at the same time to report a few cases, which may prove of some interest.

This special form of functional trouble, not due to alcohol or tobacco, is by no means, a common one; this is especially true in regard to the cases of amaurosis; those of amblyopia and narrowing of the fields of vision, being more frequently met with.

The onset of the disturbance is usually sudden, and generally follows some shock, either mental or physical. In attempting to examine the eye there is a spasmodic contraction of the orbicularis muscle which is increased on exposure to a bright light, at the same time causing a sense of anxiety, profuse lachrymation and a spasmodic closure of the lid of the eye. The globe itself does not present anything abnormal, except that in a certain number of the cases, one may find some anæsthesia of the cornea. (This was a marked symptom in one of my cases, Case 4.) The pupils are equal and react readily and normally. The media, lens, vessels and fundus are normal: so that the local examination does not lead to any knowledge of the pathological nature of the other symptoms which one learns from the patient. Question



the patient and she complains of defective vision, pain either ocular or supraorbital, and great sensibility to light. There may be absolute loss of sight, generally in one eye, or only amblyopia and a reduction of the field of vision to a small area around the fixation point. Besides the above we find the local and general symptoms of hysteria, viz: A circumscribed pain over the brow, a globular sensation in the throat, excitability and irritability of the nervous system, palpitation of the heart, a tendency to laugh and cry without cause; irregular, painful or absent menstruation.

The following cases were referred to me by Dr. David Webster and well illustrate the above objective and subjective symptoms.

Case No. 1.—(Amblyopia and contraction of the fields of vision) Vision restored in twelve sances.

Kate T.—Eighteen years old, was seen for the first hypnotic sitting on the 12th of June, 1894, when the following history was obtained. She had always been nervous, but otherwise had had no trouble until the appearance of menstruation two years ago; then during the first year she suffered much pain each month. For the past year menstruation has come on at irregular intervals and has now been absent for two months. Within the last six months the patient has become very depressed and emotional, and she has also had two convulsive seizures of a hysterical nature. One month ago a brother committed suicide, and it was shortly after this she first noticed failure of vision, at times becoming entirely blind; this latter condition only lasting a few seconds. Examination does not reveal any organic lesion of the nervous system. Any attempt to examine the eyes causes a spasmodic closure of the lids. Both pupils are moderately dilated and react normally. The vision of the right eye is 20/15 and of the left eye 20/40. Both fields of vision are very much contracted.

The fundus, media and vessels are normal, nothing being found to account for the condition present. Two attempts to hypnotize the patient failed but on the third trial she passed into a deep sleep and suggestions referable to the conditions present, were made. After seven sances, the fields were again measured and a marked improvement was found.

The treatment by suggestion was continued until August 15th, when the patient was discharged with perfectly normal fields and vision fully restored.

Case No. 2.—(Amblyopia. Contraction of the visual fields. Improvement after six seances.)

March 29, 1895. Charles T.—Age twenty-eight. Single. Clerk. General health good up to eighteen months ago; then after some business troubles and excesses, he became depressed, nervous and could not sleep. Six months ago he noticed that he was having periods of temporary blindness, these only lasting for a second or two. Three months later his vision became blurred and this condition has remained up to the present time. He has never had any diplopia. Within the last month there has been more or less frontal and occipital headache, and lately he has become very emotional. Sometimes he gets confused and cannot remember dates, otherwise his memory is good. The urine has been examined several times and always found to be normal. At one time he had herpes preputialis, and being told that it was due to a syphilitic infection, thought all his symptoms were caused by this disease.

Examination. Stands well with eyes closed. Knee-jerks high but equal. Grasp of hands as shown by dynamometer—R 44-40. L-40-39. Tongue straight; speech normal. Pupils equal, of medium size and active. The ocular muscles thoroughly tested with prisms, do not show any degree of paresis. The optic nerves and retinal vessels appear perfectly normal. Both visual fields are very much contracted.

There is no loss of color perception. The left side including the cornea is slightly anæsthetic and he now states that he occasionally has a temporary feeling of weakness in the leg and arm of this side. Examination does not reveal the slightest loss of power; all the muscles are firm and react normally to both the faradaic and galvanic currents.

He was hypnotized without any difficulty and the proper suggestions were then made. This method of treatment was continued for two weeks, resulting in a complete disappearance of many of the symptoms complained of. The visual fields were again measured and though some improvement was shown, still there remained a certain amount of contraction.

On April 12th he stated he had been having more or less pain in his abdomen for several days, and that this morning he had passed some white objects at stool. Upon examining these, they were found to be a number of the links of *tænia solium*. A mixture of castor oil

and felix mass was ordered, with directions as to diet, &c., which resulted in the passing of two worms, many yards in length, including the heads. Three days later his eye symptom had disappeared, the visual fields were entirely normal and have remained so up to the present time.

Case No. 3.—(Monocular blindness. Cure in five Seances.)

Fannie T.—Thirty-eight years old. Married. Seen November 23, 1892. She was perfectly well until one month ago, about which time she became depressed, could not attend to her household duties and had a number of crying spells. Three weeks later pain appeared in the left eye accompanied by some blurring of vision, which gradually increased and now for the past three days there has been complete blindness in this eye. No vomiting, diplopia or vertigo. There is no history of rheumatism, malaria or any serious illness, having always enjoyed good health up to the time of the present trouble. She has had three healthy children and has had no miscarriages. Careful inquiry does not reveal any history of syphilitic infection. Bowels and menses are regular.

Examination.—There is a complete loss of vision in the left eye; she fails to recognize any object held before it, the right eye being covered. Vision in the right eye normal. Pupils normal in size and active; no ocular paresis. On exposing the eyes to a bright light there is a certain amount of photophobia. The ophthalmoscope shows nothing abnormal either in media, disc, or vessels; with the exception of a slight amount of anæsthesia of the cornea, there were no other sensory changes. The patient was easily hypnotized, and the proper suggestions having been made, she was awakened after sleeping ten minutes. The right eye was then covered and the left again tested. Vision was about the same, but she volunteered the statement that there was less blurr, and the pain had disappeared.

November 25th. No change; condition the same. Vision again tested as before and with the same results. She complains of a good deal of pain in the left eye and face. I hypnotize her and suggest that she will have no more pain; that the rest will do her good and that she will now see. In fifteen minutes she sits up on the couch and placing one hand over the right eye, recognizes a clock on the wall twelve feet distant, although

she is unable to distinguish the hands or tell the time. A bunch of keys, a knife, and a silver half dollar are named correctly at four feet. The pain is entirely gone.

December 7th. Patient reports that she is now able to see much better, also that the pain has been absent since the last seance. She sees well at ten feet, but beyond this everything looks blurred. Seance repeated.

December 9th. Has been perfectly well; no pain. Vision entirely restored.

Case No. 4.—(Monocular blindness. Cure in nine Seances.)

On September 12, 1894, the patient was referred to me by Dr. Webster with the following note: "I see no sufficient ophthalmoscopic reason for the loss of all but perception of light of this young woman's left eye. If you can find no lesion of the brain to cause the blindness, perhaps you can restore the sight by hypnotism as you did before."

Mary C.—Eighteen years of age. Married. The patient is an Armenian and cannot speak a word of English, but her physician is present and acting as interpreter, the following history is obtained: Has been married five years; two children and no miscarriages; is now nursing an infant eight months old. Was fairly well up to the past month; during this time she has become depressed, emotional, and has often complained of a lump rising in the throat. A few days ago she had some head pain and with its appearance she noticed failure of vision in the left eye, and now the patient cannot distinguish any object at any distance far or near, but she can make out the difference between light and darkness. She is poorly nourished and anæmic. Mucous membrane of lips, gums, and conjunctivæ pale. Both pupils moderately dilated, the left not reacting as actively as the right. Fundus entirely normal. Failing to find any evidence of organic trouble, the diagnosis of functional amaurosis was made, and I decided to try suggestion for its relief. The patient not being able to understand English, I was not successful in my first attempt to hypnotize her, but through the aid of Dr. Attarian I learned the proper Armenian words necessary for the purpose, and was successful at the third seance in obtaining a deep hypnotic sleep. Complete recovery took place in nine seances.

Case No. 5.—(Monocular blindness. Unimproved.) This patient had been under the care of Drs. Boynton

and Palmer at the Ophthalmic Hospital, and was finally referred to Dr. Webster, through whose kindness I saw the case on February 4th, 1895. Although there does not seem to be any doubt in my mind as to the trouble being a functional one, all methods of treatment have proved ineffectual. The following is the history.

Agnes L.—Fourteen years of age. Single. She was perfectly well up to one year and a half ago; about this time she fell on the ice, striking the back of her head. She was much frightened and jarred, but did not vomit or lose consciousness. Menses appeared for the first time shortly after this, but have never been regular, two months having now elapsed since the last period. Two months after the accident above referred to the patient commenced to have headache, chiefly frontal, but sometimes in occiput and back of neck. On May 5th, 1894, she came under the care of Dr. Boynton at the Ophthalmic Hospital, for dimness of vision. Examination revealed almost total loss of vision in the right eye, without any change in the fundus, vessels and disc, all being found normal. While in the hospital she had two epileptoid attacks, hysterical in character, and she also had frequent crying spells. Thorough treatment with strychnia, glonoïn, iron and valerianates caused no improvement.

Examination.—There is a complete loss of vision in the right eye. Left eye vision 15/200, brought up to normal with proper glass. Field much contracted in all directions. In a moderate light both pupils dilated, the right being a little the larger. Reflex through the right retina not as good as through the left. When one illuminates the left retina, the right pupil does not remain contracted as long. With direct illumination the reaction is about the same. There is a marked loss of sensation of the cornea of the right eye, but no anæsthesia of face, body or extremities. The patient recognizes colors readily. The knee jerks are exaggerated but equal. The question of simulation was considered, but repeated tests by prisms and other means gave negative results. Treatment by means of drugs having failed to change the conditions present, hypnotism and metalotherapy were tried, but both failed to influence the patient in any way.

Prognosis and Diagnosis.—From the facts brought out in the above histories we see that an amblyopia or amaurosis from hysteria may be slight in form and tran-

sitory in duration, or very severe; sometimes tardy in its progress and prolonged in its existence. If the condition should persist for any length of time, and an alteration of nutrition or any morbid formative process be set up by prolonged functional disturbance, then it is probable, that finally the condition would change into an amaurosis from inflammation and congestion. This latter condition, I now believe, is taking place in case 5, which illustrates well this type of the disease.

The diagnosis is based on two points, viz: the absence of any demonstrable changes in the eye, and the lack of that agreement between the individual symptoms constituting the disturbance of vision, which under other circumstances they would exhibit. Persons whose visual fields are unusually contracted, still move with perfect security, without stumbling in a place which is not well known to them. The only difficulty in diagnosis exists in those cases where a line must be drawn between true stimulation and hysterical blindness, that is one having an actual existence in the imagination. It is not that they will not, but they cannot will. The retina receives the impression, but through some fault of the higher cortical centers, perhaps by inhibition, the patient remains unconscious of it.

Treatment.—Besides the measures usually recommended in the treatment of these disorders, the internal administration of strychnia, iron, etc., I wish to urge the trial of hypnotism, and I do not do this from any optimistic point of view. A great many of us are prone to look upon this entire subject as either belonging to the domain of quackery, or believing that it requires some special power, and thus hesitate to take advantage of this method of treatment. The time has now passed for any such argument, and any physician who would take the trouble to study the subject, would obtain satisfactory results in a certain number of cases. The manner of procedure in producing hypnosis is given in detail in current literature, so that it does not seem necessary to dwell upon this part of the subject here except to state that the fixation method is the one generally employed.

From the result obtained in the cases just submitted, the following conclusions may be drawn, viz:

1. We possess in suggestive therapeutics an important aid in the treatment of certain morbid conditions, but just how valuable this may be cannot be estimated, until it is more generally used and the results reported.

2. The results of this method of treatment is sufficient to stimulate the profession to further use of it. 3. Instead of waiting and trying other methods first, thus allowing the disease to exist for a certain time, I would recommend the trial by hypnotism in the first place. Two of the cases already reported have been treated by other measures for some time without success. 4. The use of hypnotism by the intelligent physician in the cure of certain morbid conditions does not produce any bad effects, notwithstanding reports to the contrary.

## DISCUSSION.

Dr. DERGUM, of Philadelphia. I infer from the account given that in all these cases color perception was normal. That is an exceedingly interesting fact when we take into consideration the statement of some foreign and American observers that the color field in hysteria is very often reversed. In my own experience the reversal is not as common as is generally supposed. In the past year I have had every case of hysteria that came under my care at Jefferson Hospital carefully studied with regard to this point, and I did not see a single case of reversal of the color field, so that in my own experience, at least, this symptom of hysteria—reversal of color field—does not occur as often as we have been led to think.

Dr. PRINCE, of Boston. I should like to ask Dr. Booth whether he made any tests while his patients were hypnotized with the view of ascertaining whether the patient really recognized objects which she claimed not to have seen in the waking state. It will be remembered that it has been shown that some hysterics, some of these people with apparent blindness, are not really blind at all in one sense of the word. Binet, Janet and other French observers have shown that if you hold up before one of these hysterics with amblyopia something which he says he cannot see, and afterwards hypnotize him, he will tell you everything you exhibited before him when he was apparently blind. In other words these patients really do see. It is not a real blindness. Whether this is universal with all hysterics may be questioned. I doubt if it is so, but it is true of a certain number. In other words, the blind hysteric sees, the deaf hysteric hears, the hysteric with loss of sensation feels. This I have been able to demonstrate

myself, so far as anæsthesia is concerned, in two cases. In one of these cases, for example, I was surprised to find that the patient who had lost sensation in the right hand not only recovered sensation in the hypnotic state, but in this state could describe accurately what was previously done to the anæsthetic hand while she was in the waking state, even to stating the exact number of times it was pinched with a pin. When I put a pencil, a piece of paper, a little piece of rubber, a pair of scissors, etc., in the anæsthetic hand, she not only failed to recognize the objects, but even to perceive the contact with the skin; yet after the subject was hypnotized she told me everything I had done, and named correctly the objects. These experiments confirmed what has been found by others. Now I am quite certain the anæsthesia was real and not the result of unconscious suggestion or feigned, because at the time I was unprepared for the results. In other words, the hysterically blind and anæsthetic see and feel, but their visual and tactile sensations are not in connection with their dominant waking consciousness.

This has led to a very interesting and plausible theory of the pathology of these cases. It seems as if there was a shutting off from the field of consciousness of certain perceptions which formed a group of segmented sensations more or less by themselves, and which by artificial means (hypnotic state) could as memories be brought back into relation with the remainder of the conscious field.

There is another very interesting phenomenon connected with amblyopia which, I believe, has been established by careful and competent observers, although the nature of the phenomenon is such as to excite skepticism. It will be remembered that Painaud pointed out an extraordinary fact namely, that an hysteric with monocular amblyopia is blind in the affected eye only so long as the sound eye is closed, but that when both eyes are open vision returns in the blind eye, that is, he has normal *binocular* vision, but *monocular* blindness. These observations of Painaud have been confirmed by Pitres, Charcot, Bernheim and others. I have had an opportunity to demonstrate this phenomenon in a case of traumatic hysteria. All the usual tests to detect malingering were used, and although it is not possible to so present the evidence that it will have the same weight with others as with those who made the examin-



ation, the tests and circumstances were conclusive in our mind that we have to do with a *bona-fide* phenomenon. The phenomenon is an important one, as it shows that the ordinary tests to determine the simulation of organic blindness are not applicable to hysterical amblyopia. I hoped to be able to exhibit this patient before this Society to day, but his mental condition is still such that it is not possible to do so. The case I refer to (for the opportunity of examining which I was indebted to Dr. E. E. West, of this city,) had monocular blindness to the extent that when the right (sound) eye was closed, he could see with the left eye a candle flame only as splashes of light, not as a distinct flame. When a prism was held before one of his eyes, he not only saw double, but two distinct flames, identical in every respect. Further, with the ordinary tests for simulation, the screen test, the prism test, the color test, this man had perfect binocular vision. Monocular amblyopia was present only when the right eye was closed. These are very important observations, and although there is need for this further confirmation, still the evidence thus far furnished by a number of competent observers is so strong that I believe the phenomenon case be accepted as a fact. I wanted to ask Dr. Booth whether he had made similar tests in his cases. I doubt very much whether this peculiarity of hysterical amblyopia is found in all cases; in fact, Painaud did not always find it, and in one case I failed to obtain it.

How common it is remains to be determined as well as the pathological explanation.

Dr. BOOTH, of New York. The patients were thoroughly tested for binocular vision by Dr. Webster and myself, and in the cases of monocular blindness they all showed that they did not have binocular vision with the prism test.

Dr. WALTON, of Boston. The point to which Dr. Prince has called attention, namely that some patients with monocular blindness can see with the blind eye when called on for binocular vision, is one of great interest, and I should quite agree with him as regards the probability of hysterical amaurotics really seeing more than they realize, that they do so through lack of activity of the higher centres. Still, when we have to do with medico-legal cases it is not sufficient demonstration of genuineness to prove that the patient sees what he claims he does not. There is a link wanting in the chain of evidence

here even if we acknowledge that he is not *necessarily* simulating, but may be a victim of hysteria. It seems to me we are in danger of regarding cases as genuine hysteria because they follow certain types, whereas the variations of hysteria are so great that hardly an anomaly can be imagined which might not be included under this diagnosis. For example, none of Dr. Booth's cases saw with the blind eye when binocular vision was tested by prisms, a condition the exact converse of that described by Dr. Prince.

Dr. BOOTH, of New York. I have nothing to say in closing except that I was led to report these cases on account of the interest I took in them and the results obtained. The remarks last made in regard to simulation, etc., refer more especially, I think, to accident cases, railroad cases in which there is some reason and cause for simulation. The cases I reported were not of that class. They had no reason for simulating, although thorough testing was made by several of us for that very purpose.

## A CASE OF COMPLETE HYSTERICAL ANÆSTHESIA IN THE MALE.

BY GEORGE J. PRESTON, M.D.,

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LOSS or alteration of sensation is certainly the most constant and characteristic of the hysterical stigmata. The sensory disturbance is not confined to the skin, but involves likewise the mucous membranes and the special senses. Again, all the component parts which go to make up sensation may be involved, or only certain of them. The degree of sensory disturbance varies from slight blunting of perception up to the point where no peripheral irritation is appreciated. Hemianæsthesia is the most characteristic form of hysterical anæsthesia, but is not, in my experience, nearly so common as the irregularly disseminated form. Total anæsthesia is certainly very rare. Briquet, in his classic collection of cases, met with it but four times. Szokalsky, Heyne, Charcot, Gilles de la Tourette and others have mentioned a few cases. Of course, it is necessary to restrict the term to cases that last for some time, and not to include those cases in which there is a mere transient loss of sensation over the entire body, since this latter condition is not uncommon. The following is the only case of total hysterical anæsthesia that I have ever seen:

Frank E., aged 31; a plasterer by trade, was admitted into the city hospital December 27. Family and personal history unimportant. Is a fairly temperate man, though at times he drinks a little too much. On Christmas night he had been drinking, and during a row was kicked in the face. When admitted to hospital he had some slight ecchymoses about his face. His breathing was very rapid for a day or two, though there were no physical signs of any trouble in his lung. The day after he entered the hospital it was discovered that he had a patch of anæsthesia about the size of a dollar in the left parietal region. There had been no injury to this part of the scalp. On January 6 the patient developed a gen-

eral anæsthesia. Tactile and pain sense were entirely lost, even the cornea was insensitive. Temperature sense was entire gone, and muscular sense impaired but not lost. The patient was not able to distinguish between quinine and sugar. Tested with various odors he was entirely unable to distinguish between them. Hearing was impaired, though he says he was slightly deaf before. There is marked constriction of the visual field, and reversal of the color fields. Reflexes superficial and deep were normal, except that the superficial reflexes were perhaps sluggish. Electric reaction was normal. On January 28 the patient was hypnotized, and while other suggestions were potent, for example, he was made to vomit by suggesting to him that he had drunk ippecac instead of water, the suggestion that his sensation had returned had no effect. After this he was hypnotized regularly several times a week and the suggestion strongly made that sensation would return. By the middle of April marked improvement had taken place; he began to regain both tactile and pain sense, and the special senses began to return. For example, he had not noticed whether he had sugar in his coffee or not; he now began to complain that his coffee was too sweet. The visual field began to clear up, as can be seen from the diagram made for me by Dr. Harry Friedenwald. At present he has regained his normal sensation in all respects. There was never at any time any suspicion of the genuineness of the case. The man was anxious to get well, and while he was in the hospital was a most efficient worker.

Of all the tests to distinguish spurious from true hysteria, I consider the careful examination of the visual field by far the most valuable. It is practically impossible for any patient to simulate the well-known changes in the visual field, if repeated examinations are made and recorded. This symptom, so common as Dana has pointed out, should be more generally employed than it is, since it constitutes an almost certain test.

#### DISCUSSION.

Dr. GEO. W. JACOBY, of New York.—In consequence of a peculiar case of total hysterical anæsthesia seen by me last winter, I had occasion to look up the literature of the subject for the purpose of determining the frequency of such occurrence, and was astonished to find

that total anæsthesia is considered rare. I have seen a number of such cases, the majority occurring among the insane, patients suffering from paranoia or acute hallucinatory insanity. The case to which I desire to refer particularly was a girl with loss of sensation over the entire surface of the body, loss of hearing, loss of taste, but without affection of sight. Cases of this kind should cast some light upon the mode of production of ataxia, but my patient, even with closed eyes, showed absolutely no sign of such disorder. I should like to ask Dr. Preston whether in his case any ataxia was present. Another interesting question is the one which was raised by Strümpell in a similar case; it is as to the cause of sleep production. Strümpell's patient upon closing the eyes and occluding the ears with cotton, thus casting off all external excitations (none being conveyed by the skin) would at once go into a sleep-like or hypnotic state; in my patient and in similar cases described by others, this experiment could not be successfully repeated. Diagnostically, I should like to say that in the majority of cases of hysterical anæsthesia, whether complete or incomplete, while the skin itself is perfectly analgesic, painful points can usually be made but upon deep impression. It is this presence of pain to deep pressure with its absence to all forms of skin irritation which is so characteristic of hysteria.

Dr. WALTON, of Boston.—If I may be pardoned for again alluding to the question of genuineness of hysterical cases, the remark has been made, in this connection, I believe by Dr. Riggs, that a certain patient could not be simulating, as the visual fields taken at different times always corresponded. This is in line with the view already advanced that constancy of anæsthetic boundaries, pressure on the dynamometer and similar tests establish genuineness in claimants for damages. I am not prepared to accept this dictum. When we consider the variations of anæsthetic boundaries, dynamometric pressure and visual fields often found in genuine cases, variations for which we must make due allowance before we declare a claimant a fraud, we should not be too hasty in regarding a case genuine because his answers come within these limits, nor even necessarily because he maintains the greatest exactitude. My own tests would lead me to believe that an intelligent and practised simulant could maintain even greater constancy in some of these subjective tests (for example, the dyna-

monometer) than the average genuine patient is likely to do.

Dr. W. M. LESZYNSKY, of New York, said that with proper precaution during the examination, and with our present methods of investigation faithfully and persistently carried out, it was impossible for the cleverest patient, even an accomplished ophthalmologist, to simulate successfully defective visual fields.

Dr. PUTNAM, of Boston.—Perhaps the discussion has wandered too far, and I shall not detain the society long. This point of Dr. Walton's is real and practical, and unless the chairman rules it out of order I should like to say a single word about it. Of course, no one who studies these cases much would maintain that the clinical picture is an absolute one. There is no doubt that the hysteric symptom-complex varies from day to day as the circumstances vary. It is for us to detect the law of variation, as from fatigue, etc., and to train our instincts so that we may be able to substantiate the information we derive from these tests by other signs which we cannot express so well. When we get up in court and say the hysterical patient does this or that, and, therefore, is hysterical, or that a certain person is a simulant, we give as well as we can the reasons for making us think so, but I do not think we are bound on that account to deny that no other person could come fairly near the same test, or bound for that reason to reject the diagnosis to which our instincts and training have led us. The reasons we give the jury from, of necessity, only a part of those which guide our judgment. Having seen vast numbers of these cases, and putting the accurate or comparatively accurate information we get from these tests together, with what we know of the laws of hysteria and the laws of the person in health, we do form, it seems to me, in almost every case an accurate judgment.

Another point I think is important, and that is that persons in health, of course, are what one might call hysterical to a certain degree. One sees that in court, with regard to testimony in questions of observations and other matters. A great part of the peculiarities of the field of vision in amblyopia are due to variations in attention, and one sees evidences of that attention or lack of attention in daily life. You see persons testifying to something they observed, saw or heard on a certain occasion, and the question is often asked, why they

noticed the fact testified to on that day when they would not ordinarily notice it. In reality it is an affair of retro-active attention. And so also with the hysterical persons Dr. Princee referred to. It would be ridiculous, it seems to me, for an expert to try to seek a hard-and-fast rule, to try to prove all the time that his own judgments were wrong because such apparent discrepancies arise. If a person claims to have seen a thing which in all probability he would not have noticed, we must judge from every thing taken together whether he saw it or not.

Dr. GRAY, of New York.—The wider subject that Dr. Putnam has introduced would be more proper under the discussion on the next paper. I move that we pass on to the reading of that paper, and then we can if we choose do justice to both subjects.

Dr. PUTNAM, of Boston.—It seems to me before putting Dr. Gray's motion there might fairly be opportunity for discussion upon Dr. Preston's paper on total hysterical anæsthesia, if there are any further remarks upon that.

Dr. W. A. JONES, of Minneapolis.—I want to ask Dr. Preston whether in his case of total anæsthesia and anæsthesia of the eye-ball and cornea, he found immobility, and whether such things may not be present in purely hysterical cases,—immobility of the eye-ball with total anæsthesia of the cornea.

Dr. TAYLOR, of Boston.—Having seen a case of total anæsthesia myself, to which I do not propose to refer, I should like to ask if in Dr. Preston's case there was difficulty in locomotion under excitement more particularly. My case was mistaken for locomotor ataxia.

Dr. PRESTON, of Baltimore.—There was no disturbance of motion in this case. The muscular sense so far as I could tell was only slightly impaired. The man possessed a relatively good idea of the position of his limbs, while not a perfectly accurate motion, still relatively good. I should say that his muscular sense was not impaired. There was not the slightest ataxia, or any disturbance of motion.

While I call this the only case I have seen, I have seen two cases somewhat similar to the ones Dr. Jacoby alludes to, one clearly insane in which there was total anæsthesia, and another case which I must confess I have not made up my mind whether there was any lesion or not; whether the anæsthesia was purely hysterical or not.

Just to touch for a moment upon the point Dr. Walton has raised in regard to the importance and usefulness of the visual test with limitation of the visual field, I think that indisputably it is the best test we have for malin-gering. I do not think it can be successfully simulated. Of course, anything can be simulated. We know that the simulator may deceive the very elect, but it seems to me, it is by far the best test we have for hysteria. While I do not mean to say that there may not be here and there successful cases of simulation of the visual field, I do mean to say if the tests are carefully made with the perimeter and records made of them, and thus obtain a sort of composite of the visual fields, I think it is as nearly a perfect test as we have. I agree with what Dr. Putnam has said, that we are to judge of hysteria and make our diagnosis by the *tout ensemble*, not pick out a symptom here and there, not pick out a symptom and say: "This is an absolute symptom of hysteria, and this may be simulated." It is the general aspect of the case. To those who are specialists it is the general impression the case makes on us and not any single symptom or any single set of symptoms. We are too apt to forget that after all hysteria has nothing to do with the peripheral organs. In regard to the eye, sensation, etc., we are always obliged to bear in mind that it is the central and not the peripheral organ that is affected. The lens may be all right, but it depends much on the condition of the background, as on position of the lens as to the image we get; so that while the peripheral organs may send in all sorts of stimuli, they are received and interpreted in very different ways. While we are ignorant of the true pathology of hysteria, whether we consider it a distinct nutritive disturbance of certain cortical cells or not, we do know that the disturbance is more or less transient, that under the influence of certain strong stimuli the cells may be made to functionate. The cell may very rapidly regain its power, so that in considering the genuineness of the various symptoms I think we are liable to regard the peripheral rather than the central organs. There was no immobility of the eye-ball at all. The cornea was absolutely insensitive, but the examination revealed no abnormal condition.



## TWO CASES OF "RAILWAY-SPINE" WITH AUTOPSY.

BY F. X. DERCUM, M.D.,

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THE opportunities for autopsies in the class of affections grouped under the head of "railway spine" are so infrequent that it seems important to place the following cases upon record.

CASE I.—In this case there was no element whatever of litigation, and it is, therefore, of very great value. It was in part reported by the writer in a paper on "The Back in Railway-Spine," read before the New York Neurological Society, on May 5, 1891, and later more fully in a paper entitled, "A Case of Railway-Back," (*JOURNAL OF NERVOUS AND MENTAL DISEASES*, January, 1892). This record is herewith briefly reproduced.

G. T., aged forty seven, single, and an upholsterer by trade, was in good health up to October 22, 1890. On that day he was sitting on the rail of the South street bridge (Philadelphia). His hat blew off, and letting go his hold upon the rail to catch his hat he lost his balance and fell a distance of some thirty feet upon a mound of earth. He struck upon the back and head, became unconscious and remained so until he found himself in the University Hospital, to which he had been removed on the same day. He was at first very much confused and suffered intensely from pains in the back, and his entire body seemed to tremble. On October 27 he was discharged, and the same day admitted to my wards at the Philadelphia Hospital. When first seen by me, he walked into the office of the nervous pavilions, walking without assistance. He seemed, however, weak, and his steps were evidently shorter and slower than normal. He stripped to the waist without help. He complained of pain in the lower dorsal and lumbar regions, and here, deep pressure revealed great soreness. Marked pain was also elicited in this region by flexion, torsion

and transmitted shock. Marked spasm of the muscles in this region was also noted on movement. In addition, there was marked tremor of both arms and shoulders. He also complained of headache and seemed much depressed.

He was at once placed in bed on the rest cure. Milk in as large quantities as he could take was given, and for a time, massage was attempted, but this had soon, owing to the painful condition of the back, to be abandoned. Instead of improving, his symptoms steadily increased in severity. His back became more and more painful. The muscles soon attained a condition of almost constant spasm, and as a consequence, rigidity was very marked. The back soon became sensitive to superficial pressure. Excessive sweating also set in. Tremor became more pronounced than ever. Four weeks after admission, his symptoms had attained their height. The man was thoroughly and abjectly miserable. He was excessively depressed, cried easily, complained of headache, said that he could not sleep, dreamed sometimes that he was falling again from the bridge, had ringing of bells and hissing noises in his ears, trembled worse than ever, had difficulty in passing his water, frequently had sharp pains shooting through his back and head and even in his abdomen. In addition, there was now decided loss of sensation in both feet and he was utterly unable to stand. His weakness was extreme. The sweating continued unabated; bowels were constipated; knee-jerks much exaggerated; micturation frequent.

He remained in this condition with but little change until the latter part of February, 1891. The spasm of the muscles was now wide spread. Originally, it will be remembered, this spasm had affected only the muscles of the lower dorsal and lumbar regions. Now it radiated to all of the muscles of the back and even of the shoulder.

His speech was at first short and jerky, and at times it was so interrupted and spasmodic as to resemble that of a patient suffering from a chill. Efforts at speaking seemed to increase the spasm of the muscles in his back, and fatigued him very much.

In order to secure absolute rest for a time, a plaster jacket had been applied. In the latter part of February, this jacket, which was worn about a month, was removed. The patient now passed from my hands to those of my colleague, Dr. Sinkler, who again instituted massage.

The latter treatment was now well borne and appeared to be followed by a more marked improvement. However, the patient lingered in the wards until the following June, when he left the hospital, walking out without the aid of crutches.

October 21, 1891, he was again admitted to my ward, having suffered some exacerbation of his symptoms. After leaving the hospital he had rested with some relatives at Ridley Park. Here he seemed to steadily gain in strength until the later part of July, when, after too great effort at walking, the pain in his back again grew worse, the tremor very much increased, and he lost considerably in weight.

His condition at the time was as follows: Entire trunk rigid. Spasm of muscles very marked over all the muscles of the back, shoulders and chest, and even noticeable in the muscles of the arms and thighs. Over the back and shoulders they were hard and firm. Here and there, spasm of individual muscular bundles simulated fibrillary contractions, more noticeable in the deltoids than elsewhere.

The spasm was markedly increased by attempts at flexion of the trunk either forward or lateral, as well as by torsion. At the same time the patient complained of pain in the lumbar and lower dorsal and cervical regions. Pain was also elicited in these regions by transmitted shock and deep pressure. The spasm of the muscles was increased by percussion, but there was no hyperæsthesia of the skin.

Almost as striking as the muscular spasm was the excessive tremor which was marked in the head, arms and legs. It was coarse and of wide extent. If the patient lay down it was diminished, if he exerted himself it was increased, though he could momentarily lessen it in the hand on attempting to grasp an object. Evidently this tremor was in some way related to the profound disturbance of the muscles so typically seen in the back.

The man walked slowly and with difficulty, both by reason of weakness and of pain. The knee-jerks were much exaggerated and there was paradoxical contraction of the tibialis anticus.

There was no loss of sensation in the legs. Frequent micturition was, as before, a marked feature, the man being compelled to rise several times at night. Sweating was still excessive though less so than for-

merly. Sleep was still very bad, the patient waking frequently and very suddenly. However, frightful dreams and night terrors did not occur as they did formerly, and in this respect the patient was better.

He still suffered from severe occipital headache, the pain extending forward in a line with the base of the skull to the brows. Tinnitus aurium, formerly very pronounced, was present at times.

He still spoke with difficulty, though his speech was less jerky than formerly. He remained at the Philadelphia Hospital with the exception of a few months each year which he spent at Ridley Park with a sister, almost continuously until the spring of 1894. During 1892 and 1893 his condition had undergone little or no change, save that he not infrequently became involved in difficulties and quarrels with neighboring patients in the ward and was given to occasional outbursts of anger which made him at times almost uncontrollable. Physically he was weak and spent his time either in bed or in a rolling chair. Mentally he was depressed and hypochondriacal in the extreme, having long given up all hope of recovery.

In the early spring of 1894 he finally left the hospital presumably to again visit his sister. Some weeks afterward word was brought that he had died quite suddenly in the Cooper Hospital in Camden, his death being attributed by the hospital physicians to acute alcoholism.

In this case the symptoms may be briefly summarized as follows: Excessive sprain of the muscles of the back and of the trunk generally, with marked spasm of the muscles and tremor together with increased reflex excitability of the muscles and tendons. In addition, the symptoms so commonly observed in traumatic neurasthenia were typical, namely, disturbed sleep, startling dreams, sudden awakening with fright, excessive sweating, frequent micturition, occipital headache, tinnitus aurium, marked general weakness, etc.

The autopsy was made by the writer, with the assistance of Dr. J. H. W. Rhein, in Philadelphia, some thirty-six hours after death. No gross changes were found at the autopsy in the nerve centres or in any of the viscera, save slight fatty infiltration of the liver, a slightly granular condition of the kidneys and a few patches of beginning atheroma in the larger blood-vessels. The brain, spinal cord, medulla and pons, the median, ulnar

and sciatic nerves were removed for microscopic study. This study was afterwards completed, the Weigert, carmine and hæmatoxylin stains being employed. As might almost have been expected from the "functional" character of the symptoms, no changes to which any significance could be ascribed were found. The sclerosis of the smaller vessels mentioned by some writers was not revealed, nor were changes noted in fibres or nerve cells which could not be attributed either to the length of time after death at which the autopsy was made, or perhaps to the methods of preparation. To the interpretation of these negative results we will presently return.

CASE II.—H. H. D., aged forty-six, a contractor by occupation, was riding in a sleigh on January 15, 1893, at 10 P. M., and while endeavoring to cross a railroad was struck by the engine of an express train running at forty miles an hour. He was tossed high up in the air so that the engine and its tender passed under his body which in its descent struck the roof of a passenger car and thence fell to the ground. He was unconscious when picked up and was taken to the Episcopal Hospital of Philadelphia. Various lacerated wounds of the forehead, scalp and nose were found, together with numerous bruises about the trunk, especially about the left shoulder. Some hours after admission he recovered consciousness, and it was then noted that he had little or no power in the left leg or arm. He remained at the hospital a number of weeks and was finally removed to his home, continuing, however, under the treatment of one of the hospital physicians, Dr. Ferguson.

The writer was called in consultation February 13, 1893. It was noted at once that there was marked flattening of the left supra and infra-spinatus muscles and atrophy of the left deltoid. The left arm hung almost uselessly at the side, motion at the shoulder and elbow being restricted and giving rise to pain. The left hand and fingers presented a smooth and glistening appearance. Pressure over the brachial plexus revealed it to be exquisitely sore. There was also tenderness on superficial pressure over the spine, in the mid-dorsal and the mid-lumbar region. Flexion forward caused pain referred to the mid-dorsal region as did also flexion to the right. In addition, there was also noted a slight paresis and recurring spasm of the muscles of the right half of the face. Measurements of the arm were as fol-

lows: Circumference of the arm, right,  $8\frac{3}{4}$ ; left,  $9\frac{1}{8}$ . Forearm, right,  $9\frac{1}{4}$ ; left,  $9\frac{1}{4}$ . The patient reacted well to the various tests for the tactile sense, but upon examination it was found that in the right leg and on the right half of the trunk all sense to painful impressions had been lost.

This analgesia extended up to the level of the nipple upon the right side and was fairly well defined in the median line of the body both anteriorly and posteriorly. It was also noted that the sense of temperature had been abolished on the right leg, thigh and abdomen up to the level of the false ribs. Above this level, though present, it was very much diminished. Like the analgesia, it was fairly well limited by the middle line of the trunk. The knee-jerks showed no especial change. The cutaneous reflexes appeared to be diminished. The results of this examination briefly summarized were as follows: Severe traumatic left brachial neuritis and left brachial monoplegia with atrophy of muscles about the left shoulder. Secondly, severe sprain of muscles of back. Thirdly, right hemi-analgesia extending from the foot up to the level of the nipple. Fourthly, right hemi-thermo-anæsthesia extending from the foot up to the level of the false ribs. The man was exceedingly weak physically, but mentally seemed clear and accepted his situation in a philosophical spirit, mental depression not being a marked feature.

He was again seen in consultation on May 19, of the same year, and all of the previous symptoms were again noted. The left brachial monoplegia and muscular atrophy were more pronounced than ever. In addition the man looked badly.

Examined again on November 8, 1893, it was found that a new and striking symptom had now made its appearance, namely, a remarkable and almost complete loss of voice. In addition, it was learned that the patient had had great difficulty in swallowing solids, and that of late he had been compelled to resort to an almost exclusively liquid diet. It was further noted that in addition to atrophy of the muscles of the left shoulder those of the right side had also become involved. There was now beginning atrophy in both deltoids and in both infra and supra-spinati. The loss of power in the left arm had also increased and atrophy was now evident in its muscles. Measurements as follows: Right arm  $8\frac{5}{8}$ ; left arm, 8. Right forearm,  $8\frac{3}{4}$ ; left, 8. Hands, right,

8 $\frac{3}{4}$ ; left, 7 $\frac{5}{8}$ . Right thigh, 16 $\frac{3}{4}$ ; left, 15 $\frac{1}{2}$ . Right calf, 12 $\frac{1}{2}$ ; left calf, 11 $\frac{3}{4}$ . It was also noted that the paresis of the right half of the face observed at a previous examination was more marked, as was also the twitching of the muscles. These muscular twitchings were now also noted on the left half of the face. No reaction of degeneration, merely slight quantitative diminution was found on electrical examination. The tongue when protruded was distinctly deflected toward the left. The left half also was apparently smaller than the right; taste was apparently lost on the anterior two-thirds of tongue on either side, and certainly diminished in the posterior third.

The other sensory symptoms noted at previous examinations, *i. e.*, the hemianalgesia and hemianæsthesia, were found to be present as before, not having undergone any change. However, there was now found in addition distinct diminution of the tactile sense corresponding more or less closely with the area of hemianalgesia. The knee-jerks were now slightly diminished; there was no ankle clonus. There was not at any time any affection of the sphincters.

Pupils are small and react feebly to light, but readily to accommodation; urine normal.

On November 13, 1893, he was examined laryngoscopically by Dr. J. Solis-Cohen, who made a diagnosis of left hemi-palsy of the larynx. His report is herewith appended: "The left vocal band was completely paralyzed in a position just within that usually seen in the cadaver, its border so depressed that it looked broader than when in the normal position, and partially applied towards the external portion or wall of the laryngeal canal. Its aryepiglottic fold was tenser than that of the right side.

The right vocal band reached the middle line in phonation, but on a higher plane than the position of the left band, and its supra-arytenoid cartilage passed to the inside of the left one.

The right vocal band was unable to reach the left one, and hence there was complete aphonia. In the opinion of Dr. Cohen the paralysis began in the posterior crico-arytenoid muscle, and that, therefore, the voice remained good until the paralysis of the recurrent became complete.

Inasmuch as the paresis and the twitching of the facial muscles and the slight wasting of one side of the tongue suggested nuclear disease, it was thought prob-

able that this hemi-palsy of the larynx was nuclear in origin. However, thoracic aneurism was considered as a possibility, but physical examination failed to reveal any symptoms.

On January 28, 1894, the writer was again summoned and the following observations confirmatory of the previous examinations were made: Right facial atrophy more evident than at previous examinations; twitching of facial muscles somewhat less marked. Marked atrophy also of left sterno-mastoid and sterno hyoid and thyroid group; supra and infra-spinatus muscles markedly atrophied on both sides; rotator muscles of humerus on left side weak; gluteal muscles on both sides also atrophied; other muscles of both arms and legs exhibit signs of general wasting.

As patient stands the left shoulder is held somewhat higher than the right, the latter dropping apparently from weakness of the right trapezius. Legs and feet are livid, almost cyanotic; feet cold to touch. Both feet are flattened, the arch of the foot being lost, especially in the left foot due to muscular and ligamentous relaxation. There is also excessive varicosity of both thighs and legs; greater upon the left side. Knee-jerks plus, but readily exhausted. The tactile sense appears to be everywhere preserved, except that it appears slightly diminished in the right leg and right side of trunk. There is, however, decided analgesia of the right leg and right side of trunk as far as the median line and as high up as the fourth interspace. There is also thermal anæsthesia of the right leg and trunk as far as the median line and up to the level of the false ribs. Measurements: Over shoulder through axilla, left,  $15\frac{1}{2}$ ; right,  $15\frac{1}{2}$ . Left arm, 8; right,  $8\frac{1}{2}$ . Left forearm,  $7\frac{1}{2}$ ; right,  $8\frac{1}{2}$ . Left hand,  $7\frac{1}{2}$ ; right,  $8\frac{1}{4}$ . Left thigh,  $14\frac{1}{2}$ ; right,  $15\frac{1}{2}$ . Left leg,  $11\frac{3}{4}$ ; right,  $12\frac{1}{4}$ .

On March 9, 1894, the writer was again summoned in consultation. All of the symptoms noted previously were again observed. In addition, however, an effusion in the left pleural cavity was diagnosticated and the presence of fluid was confirmed by means of an aspirator. The number of respirations had become decidedly increased. The pulse was rapid and weak, while the heart's action was decidedly irregular. The physical signs of aneurism were sought for but without avail, all sounds being deadened by the pleural effusion which was apparently very large. He was again seen



on the following day, when his condition had not changed. Two days later, March 12, he suddenly died. Autopsy was held March 13.

Microscopic examination of the brain and cord revealed no facts of moment. On opening the cavity of the chest, however, it was found that a large fusiform aneurism, involving mainly the descending portion of the arch and of the thoracic aorta, had ruptured into the left pleura. In the latter cavity was found a mixture of serum and blood, the entire pleura being enormously distended. The walls of the aneurism, though thickened, were not markedly so, nor were calcareous deposits and other evidences of great age present. The aneurism had evidently produced effusion into the left pleura by pressure upon the pulmonary veins. The abdominal viscera revealed no changes worthy of note.

The brain, pons, medulla and spinal cord were removed for microscopic study. The methods of preparation were the same as in the previous case, namely, the Weigert method, carmine and hæmatoxylin.

The cortex revealed no change, however. Because of the facial palsy, the slight wasting of the tongue and the difficulty of swallowing it was fully expected that changes of some kind would be noted in the medulla. Here, again, I was doomed to disappointment. Changes were also sought for in the cord, especially with reference to the atrophy of the muscles of the shoulder and of the buttock, and also with reference to the hemianalgesia and hemi-thermo-anæsthesia, but here again the search was fruitless, and throughout the results of the examination as far as nerve centres were concerned was negative.

Though disappointing, the negative result of the microscopical examination of the above cases is not without significance. In the first case, we could not upon a priori grounds have expected to find decided changes in the nerve centres. The case is interesting, however, as showing the persistence for years of grave nervous symptoms without the production of so-called organic changes. It is, further, to my mind, exceedingly probable that the condition of the patient, which was the result of a trauma, was directly responsible for his death. He had been previous to his accident, as far as I could gather, a thoroughly respectable man, and though not a total abstainer, he was not in any sense of the word an inebriate, and even if the surmise of the hospital phys-

icians was correct, that his death was due to acute alcoholism, the latter can without breach of probability be fairly regarded as the outcome of his depressed mental and physical condition.

In Case II. we could, perhaps, with reason have expected to find changes both in the medulla and in the cord, although it must be admitted that the degree of atrophy observed about the face and tongue and the shoulder and pelvic girdles were not such as we are accustomed to find in well-marked bulbar palsy or poliomyelitis. The expectation, also, of finding changes in the cord to which the hemianalgesia and hemithermo-anæsthesia could probably be ascribed could not be considered unreasonable, and yet, as already stated, such changes were not discovered. This case, like the first, is exceedingly instructive as presenting symptoms in every way suggestive of central nervous disease, persisting unchanged for several years without the existence of corresponding organic lesions. It must, however, be admitted that the unavoidable delay in making the autopsy, which occurred in both cases, was such as to make it possible for minute changes, especially intracellular changes to be effaced. And, further, we have learned that the old methods of examining nerve tissue which were the ones unavoidably employed in the study of these cases, are in themselves gross methods. For many years they left us in ignorance of a world of facts revealed since by the methods of Golgi and Ramón y Cajal. I still believe that had it been possible for me to have employed these methods in the above cases, the report which I have to make would be a very different one. In other words, I am still of the opinion that changes were present in the nerve centres in these cases, especially in Case II, and that a more perfect technique, such as we have now learned is necessary, would have brought them to light.

#### DISCUSSION.

Dr. JOSEPH COLLINS, of New York.—These two papers are of extreme interest, and the cases detailed in the latter should teach us quite as much by their negative as by their positive findings. I listened attentively to Dr. Dercum's paper from beginning to end, and nothing was so impressive to me as his closing sentences. It does not seem that at this day, considering our

knowledge of the histology of the nervous system, we can rationally expect to find in such cases as those just reported anything in the brain, and in the ponto-bulbar region, with such crude methods as those of Weigert and carmine. When we have a staining at our disposal so easy of application, so devoid of elaborate technique, so ready to apply and so far superior to these as is Nissl's stain for the detection of cellular change of the nervous system, we are doing ourselves a serious injustice when we make anatomical and pathological observations without the employment of it. If we may draw conclusions without having definite premises or in any way theorize concerning these cases, and particularly the last one in which the ponto bulbar symptoms, characterized by twitchings, laryngeal paralysis were so well marked, then we are warranted in saying that a stain, such as the Nissl's method, would have shown a change in the nuclei of the cell, in the nucleoli, in the substance of the cell, or in its dendritic and protoplasmic prolongations, and the probabilities are that it would reveal them in the perivascular lymph spaces. The fact that in the second case there was such a gross anatomical change in the vascular system, which in all probability was due to the trauma, is exactly in the line in which all neurologists have been working to establish the morbid anatomy of traumatic neuroses; that is, that through slight or serious trauma there is brought about in the vascular system a change, and that the changes in the nervous system are secondary to the disease of the blood-vessels and the perivascular spaces. Our conception of the nervous system as a tree ramifying into its numerous branches, twigs and leaflets, and each one of these twigs stuck into the perivascular spaces, is a conception which is exactly in line with the supposed pathological changes. Some symptoms which Dr. Dercum has pointed out are very similar on akin to symptoms found in myasthenia gravis pseudo-paralytica as described by Jolly; in both diseases the symptoms are the result of trauma, over-fatigue or over-activity, and probably have the same ground-work or foundation.

Dr. MILLS, of Philadelphia.—It seems to me in addition to what Dr. Collins has said about the importance of finer methods in such cases, the examination should have extended to the dorsal spinal ganglia and their cranial homologues, related to sensory nerves: not only in cases of this kind, but also in various allied diseases

long supposed to be confined to the cerebro-spinal axis. As we now know the original lesion or the injury may be in these parts. It is important that they should always be examined if possible.

Dr. PRINCE, of Boston.—The importance of Dr. Dercum's paper is apparent from the evident interest it has excited amongst all present. The case which he has reported is a most valuable contribution to our knowledge of the subject. But notwithstanding the persistence of the symptoms until the fatal issue from accidental complication, I cannot help, in opposition to the opinion held by some present, regarding the whole clinical picture, as well as the findings of the autopsy, as supporting the theory of the pathology of the affection, which I believe to be the correct one. The history of this case is in entire harmony with the view that the pathological condition present was one of hysteria, and that the exciting cause was the psychical shock and not the physical shock. I don't know whether Dr. Dercum has intended to advance any theory on this point or not, but the interest of the case centres in the pathology. The negative result of the autopsy bears out this pathology. Yet I agree entirely with what Dr. Collins has said. I think that very likely if Nissl's stain had been used finer changes would have been found. But if so, what then. I do not think that any one doubts that after prolonged invalidism degenerations are liable to take place in all the organs of the body—in the stomach, the liver, spinal cord and brain; but to infer from this that these very minute changes were the original cause of these symptoms I, for one, do not believe, nor that secondary degeneration in the muscles and stomach after long continued neurasthenia is an uncommon observation. What wonder then that delicate methods might find them in the cord. Nor is it more than common knowledge that psychical derangements may induce secondary changes in the body. To my mind, the whole clinical picture of this case is evidence of a divided psychical shock from which resulted all the symptoms. As I understand the report, death was not the direct consequence of the traumatic neurosis, but of the complication, so that the final ending in no way contradicts this view.

Dr. FISHER, of New York.—I have had under observation two cases rather similar to Dr. Dercum's, but which have not as yet come to autopsy. The first case resembled almost precisely the case that Dr. Dercum

refers to, with excess of spasm. The patient had received an injury in a railroad accident, and had also recovered damages from the company before I saw him, so that that psychical element had been removed. There was nothing to be gained in any way by simulation. I examined him a number of times and always found on the slightest attempt to manipulate the muscles that the patient went into a condition of excessive spasm, chattering of the teeth and excessive perspiration broke out all over the body, the reflexes were uniformly and always exaggerated. The man, if watched at all when eating, would spill any fluid in the glass he held or was utterly unable to handle the knife or fork. When not observed he managed very well. On walking there was a great deal of spasm. Absolutely no sensory disturbances. The man certainly appeared very anxious to get well. He was dismissed from the hospital and returned in three or four months with apparently no change. This condition lasted two or three years. It does not seem possible in such a case as that, but that a minute or careful examination would reveal some effects. I cannot conceive such a functional condition continuing where the manifestations of the disease are uniformly the same without there being at the base of it some pathological change.

The second case was not unlike Dr. Dercum's in that after the traumatic injury we found complete hemianæsthesia involving the whole half of the face and the rest of the body. The patient had also lost power, immobility in connection with the anæsthesia. The diagnosis was made of a hysterical state due to trauma, and a favorable prognosis was given, which was borne out by a more or less complete recovery inside of two or three months. I would agree with the other speakers, and I should judge from what Dr. Dercum said, that there must be some definite lesion yet to be discovered in most of these cases.

Dr. BAKER, of Utica.—I somewhat regret that the paper I am to read later does not come in this group. It has some useful references, perhaps, to the subject under discussion. It has seemed to me that in observing cases which might be grouped with those reported, that we have to take into consideration this fact; namely, that at the time of accident or at the time when the first impression is made, there is set in the organism a copy, either psychical or physical, and that, as shown by some

recent studies on the part of Prof. Baldwin and Prof. Royce, and likewise by some observations incidentally made by myself, there seems to be something in the process following, by which the copy is reproduced in a series of mimeries, so to speak; and that the most serious element involved has been the matter of attention on the psychical side. It would seem to be something like this: there always results a fixation of attention, possibly upon some particular thing, or, it may be diffused, for there may be fixation of attention in a diffusible state as well as in a concentrated state. It seems to me possible that the fixation of so important a psychical matter as attention may bring about in the higher cortical centres, changes which, had we only the means of determining them, we would see to be anatomical,—the process being, first, the psychical impression; second, the series of mimeries or perpetuations of the impression; third, the resulting anatomical changes too fine as yet to be made out. I cannot get rid of the fact that in all of these cases of hysteria and other cases of psychical degeneration, and in fact in all deviations from health resulting from anything that produces a profound impression, we must take into consideration the copy set and its mimetic reproduction in a destructive series.

Dr. LANDON CARTER GRAY, of New York.—The two cases reported by Dr. Dercum form an interesting contribution to our knowledge of so-called railway spine. The more I see of traumatic cases of this kind, the more puzzled I am as to their nature. It is all very well to label them hysteria, and then assume that we know what we mean by the term, but in reality I think that we know less and less about hysteria with every year of advance in our knowledge. If you take what was thought to be hysteria a hundred years ago, and then take the forms of disease that the advance of medicine has cut off from that title, I think one can foresee that there will come a time when hysteria will die out altogether as a term; in other words, so many organic diseases that the past has not recognized have been called hysteria, so many organic diseases this day are called hysteria which are only imperfectly recognized; and I think with the advance of histological methods there will be found changes in the texture of cells in the nervous system that will demarcate even what we now call the functional forms into distinct and recognizable types under different names. I am very unwilling to

believe these traumatic cases of railway spine are pure cases of hysteria, even though we assume that there is a certain class of phenomena which we can provisionally call hysterical. In other words, these railroad cases in many instances do not answer to that conventional definition. The idea of Charcot that a concentric limitation of the field of vision, a hemianæsthesia, local anæsthesia, certain peculiar contractures were pathognomonic of hysteria, were not true of the cases we see in this country. Now the Salpetriere is a hot-bed of neurotics fostered by Charcot and his descendants up to the highest degree of neurotic perfection, and there are cases he used to hypnotize regularly for many years, and they knew beforehand precisely what they were to do much better than Charcot did, because he might forget, but they did not. Under those circumstances and when you add to that the fact that the Latin races, the French especially, are much more prone to those impressionable disorders, those variable, mobile diseases which we call hysteria, than is the composite race of this country that has come together from all the nations of the world to form a new stock, I don't think we ought to apply the deductions of the Salpetriere too absolutely. Certainly the largest proportion of the cases of hysteria seen in our clinics have no concentric limitation of the field of vision except what might perhaps be seen with very rigid examinations extending from day to day; they have no anæsthesias, have none of the so-called typical signs of hysteria. Now it seems to me you can find hysteria in almost all impressionable people, if you extend the term in that wider sense; almost all people who are impressionable are liable at some time or other to manifestations of hysteria.

Dr. DERCEM, of Philadelphia.—Both autopsies were made under circumstances which did not favor any great refinement. There was no alternative but to quickly remove the specimens and place them in Muller's fluid. I fully recognize the imperfect character of the researches based upon the Weigert and carmine stains. I am also convinced that other methods probably would have shown some changes. I think with Dr. Collins that the aneurism which was discovered was due directly to the trauma bruising the vessel wall. The force of the blow was tremendous. Doubtless the development of the left hemi-palsy of the larynx was due to this aneurism. A curious feature in this case was the fact

that the hemi-analgesia and hemi-thermoanæsthesia which I recognized the first time I saw the man, never changed, never varied in their level or area of distribution. In this connection I am inclined to question whether we are right in attributing every case of hemi-anæsthesia at once to hysteria; may it not be due to some other cause? This man was not hysterical at any time. He merely acted like a man suffering from some serious organic disease.

In the first patient there was an array of symptoms referable purely to the motor apparatus. The symptoms were essentially those which we call functional, and of course we could expect no tangible result, especially in view of the methods employed.



Wednesday Afternoon, June 5.

A LANTERN Exhibition of Photo-micrographs of Nervous Histology; Golgi Stains, was given by Dr. M. ALLEN STARR, of New York.

A LANTERN Exhibition of the Medulla Oblongata of a Chimpanzee, with other specimens, was given by Dr. EDWARD WYLLYS TAYLOR, of Boston.

EXHIBITION of the Brain of a Chimpanzee, was given by Dr. THOMAS DWIGHT, of Boston, who spoke of the brain of the chimpanzee "Jumbo," and discussed chiefly the anterior limb of the fissure of Sylvius. On the left this was distinct, ending in a bifurcation beneath a rudimentary *pars triangularis*. On the right it was represented by a minute depression not connected with the fissure.

## THE PART OF INHIBITION IN THE PHYSIOLOGY OF RESPIRATION.

BY DR. WILLIAM TOWNSEND PORTER, OF BOSTON.

In comparatively recent times the theory of inhibition has been used in a new direction, to explain certain phenomena connected with respiration. The point which is particularly in question is that of spinal respiration so-called. It is known to every physiologist that when the spinal cord is cut across between the bulb and the centres for the phrenic nerve that the phrenic nerve ceases to act apparently. The respiration of the dia-

phragm is entirely suspended, and if the animal is not given artificial respiration death follows in a few minutes. If the animal is given artificial respiration several hours it is found that on interrupting the respiration a few moments you get movements of the diaphragm resembling movements of respiration. Upon that basis the hypothesis has been raised. It is claimed that the centre from which the impulse of respiration starts is not in the spinal bulb, but in the spinal bulb and in the spinal cord; that is, that the respiratory muscle nuclei, nuclei of the diaphragm, intercostal muscles, possess the power of sending out automatic impulses and that respiration is the result of the combined automatic action of these individual groups, and not the result of discharge of automatic impulses situated in a single bulb. That is the doctrine. Section of the spinal cord causes an inhibition of the action of the phrenic nuclei, and that this inhibition lasts such a long time that no means of keeping the animal alive suffices to bridge over this period, consequently the upholders of the doctrine assume first that the phrenic nuclei are automatic centres. They support this assumption by a second assumption that section of the spinal cord causes inhibition of these centres, and if this inhibition theory does not hold good, then the original assumption does not hold good, because they cannot then explain the uniformly fatal result of the operation. It is possible, however, to show that inhibition plays no part in the physiology of respiration, at least in the part mentioned here. The claim is that when you make section with the knife across the cord that this section causes an inhibition of these cells, the origin of the phrenic cells; for so many hours that artificial respiration cannot be sufficiently prolonged to cover the whole period. The experiments which prove that this section does not cause inhibition are these. If the section is made across one-half of the spinal cord, you get in the majority of cases a standstill of the diaphragm on the same side. According to the inhibition people this standstill indicates inhibition of the nucleus of that side. If you cut the phrenic nerve during that standstill, the diaphragm on this side supposed to be inhibited begins to contract, showing that the cells were not inhibited, but merely resting. The explanation of this phenomenon is found in the fact that the respiratory impulse descends the lateral columns and is ordinarily discharged in greatest

amount upon the same side. When, however, the respiratory path on that side is interrupted, the greater amount which was discharged on that side passes over to the other side, and these resting cells which previously had received but a small portion of the respiratory impulse now receive a greater respiratory impulse and get enough to begin to contract. It must follow that two hemisections while separating the cord from the bulb cannot inhibit the respiration of the phrenic nuclei. If one hemisection did not inhibit, hemisection on the other side would not inhibit its side. Hence the inhibition theory must fall to the ground. It is certain that inhibition of respiration is not caused by a section passing between phrenic nuclei and the bulb, consequently the cause of the fatal arrest of respiration following section across the spinal cord between the phrenic nuclei and the bulb cannot be inhibition, hence the only explanation left is that the phrenic nuclei no longer receiving the impulses from the bulb and cannot originate respiratory impulses. It follows then, that the respiratory centre is not situated in the spinal cord, but in the bulb.

The general application, it seems to me, is this: I have tried to point out that the present doctrine found in the text-books is that the spinal cord contains centres for such functions as micturition and defecation, etc., centres situated in the lower part of the cord, and that these centres are, according to Goltz, inhibited by a section of the cord above them. If now the inhibition is proven not to exist for respiration, then it follows with some degree of probability that Goltz's assumption is not well grounded, and on this pure assumption rests the present teaching.

*Morning Session, June 6.*

## HYPEROSTOSIS CRANII (OPHALOMEGALY), WITH ILLUSTRATIONS.

BY DR. JAMES J. PUTNAM, OF BOSTON.

### ABSTRACT.

He reported four cases of this disease, which, to judge from published records, seems to be a rare one in its typical form, though instances of partial hyperostosis and exostosis are not very common. The most important literature on the subject consists in an analysis by Virchow in his treatise upon tumors; a monograph by Baumgarten on Leontiasis Ossea, published in Paris, 1892; a recent paper by Dr. M. A. Starr in the *American Journal of Medical Sciences*, for December, 1894.

The physiological characteristics consist, primarily, in a thickening of the bones of the head which usually occurs for the most part outwardly, though there is always more or less unevenness of the inner surface and sometimes considerable encroachment on the cranial cavity. In addition to this, an interstitial growth of the bone commonly takes place, destroying the diploe and forming a dense homogeneous tissue. In consequence of these changes the shape of the head becomes greatly enlarged, especially anteriorly and laterally. The superficial cavities and apertures of the skull are, as a rule, more or less completely filled up, so that the eyes are pushed outward and the nerves and vessels entering and leaving the cranium are compressed. The disease is chronic in its duration, beginning frequently, though not variably, in youth, and lasting often twenty or thirty years.

There are several types of the bony enlargement, of which the chief are the leonine, in which the prominences of the upper face become large and rounded, and that in which the thickening is confined to the vault of the head.

Of the cases described by the writer all belong to the latter category, except one, in which the malar

bones were prominent, but in two of the cases the orbits were more or less encroached upon.

The disease seems sometimes to originate in an inflammatory process, following injury or disease of the soft parts. Often, however, no special cause can be determined, and Virchow, while considering the affection in a general sense as inflammatory, refers to the fact that in some persons a predisposition to it seems to exist.

In Dr. Starr's case, besides the changes in the bone, there were alterations in the soft parts, such as an increase in the size of the neck due in part to enlargement of the vertebræ, and nutritive disorders of the skin, which suggested to him the possibility that the disease might be a general dyscrasia, analogous in a distant sense to acromegaly or myxœdema. Baumgarten had advanced a similar idea. In the writer's cases no distinct changes of this sort were present. Occasionally other bones of the skeleton, and, indeed, the whole osseous system show a similar tendency to overgrowth. Histologically, however, this is not to be distinguished from an inflammation, and differs somewhat from acromegaly.

The symptoms of the affection are mainly the following: Exophthalmus, deafness, blindness, facial paralysis, headache, double optic neuritis, suppuration in the ears or elsewhere, epilepsy, the attacks sometimes occurring very early, vertigo, a peculiar giving way of the leg, disturbance of respiration and mastication; loss of teeth; drowsiness; mental deterioration. Not all these symptoms are, however, present in each case.

The writer showed the photographs of three of the cases described by him, a specimen of the cranium of the fourth, and one patient was demonstrated before this Society. A case with autopsy, communicated to the writer by Dr. Morton Prince, was also read, and a remarkable skull showing the peculiarities of the disease, preserved in the Warren Museum of the Harvard Medical School, was also shown and described.

CASE I.—Female patient. No history of syphilis or other constitutional disease, except that when three years old she had caries of the ankle. It is possible that her mother and sister died from the same disease with the patient. The first positive symptom was noted when the patient was nineteen years old, though even before this she had suffered from excessive drowsiness. The

following symptoms appeared gradually and in the sequence indicated: Headache, loss of teeth, loss of hearing, discoloration of the skin. From near the beginning of the case there had been a sero-purulent discharge from both ears, and at the same time the head had begun to grow broader and the eyes more prominent. When examined by the writer, nine years after the affection began, double facial paralysis was present, and cataract of one lens. The gait was feeble and uncertain, but the hands were used freely. No sensory symptoms were present. The patient grew gradually worse, and finally drifted into the Cambridge Almshouse, where she died. For some months before her death she had suffered from chronic suppurative diffuse inflammation of the pericranium. Cerebral symptoms have never formed a marked feature of her case, and double optic neuritis was never present, so far as is known, certainly not when she was first examined. Through the kindness of Drs. Bryant and Holt the cranium was obtained for the Warren Museum. It was found to be relatively globular in shape, owing to broadening in the parietal region, and everywhere the walls were thickened, but much more anterioral than elsewhere. The forehead was prominent, and the malar processes enlarged; the orbits narrowed and shallow. The surface was everywhere uneven, but otherwise smooth. Almost all signs of sutures had disappeared. Many of the cranial apertures were narrowed or destroyed. The size of the cranial cavity was not greatly diminished, but the bony prominences were almost universally thickened and the angles rounded off. The sella tursica was smaller than normal and altered in shape. The surface of the frontal bone and the lower half of the temporals was worm eaten by caries, due to the suppurative process above alluded to, and the same process had nearly destroyed both the zygomas. The vascular grooves were practically absent, and the diploe converted into a dense homogeneous mass of bone.

CASE II. is of special interest from the fact that the disease began in infancy, the two exostoses which afterwards became so prominent, having been noticed when the patient was five years old. It is also noteworthy from the fact (alluded to also by Baumgarten) that he began to suffer from convulsions in his seventh year, before the bony growth had attracted attention. These convulsions did not recur with such frequency as to sug-

gest an ordinary case of epilepsy, though toward the end of his life, when he was about twenty years old and his disease far advanced, they became somewhat more frequent. The other symptoms consisted in dull headaches, impairment and finally entire loss of hearing, double optic neuritis of an excessively chronic course, ending after many years in blindness. Toward the end of his life he became dull and irritable. The gait was at first uncertain and then progressively feebler, so that he was finally confined to his chair. There was slight prominence of the eyes. The hyperostosis was diffuse, but was also manifested by the appearance of two large exostoses in the neighborhood of the vertex, which increased steadily in size. He died when about twenty-two years old. No autopsy was allowed.

CASE III. is that of a man, still living, who was shown at the meeting. He is fifty-three years old and gave the history that his head has been growing larger for about fifteen years. The principal symptoms have been pain, nausea and vomiting, and poor hearing. He has also been greatly troubled by a throbbing sensation in the right ear which is almost constant, unless he compresses the carotid, which relieves it. He wears a hat which is more than an inch larger than formerly, and so large that it has to be made to order. The forehead is very prominent and usually covered with moisture, and is marked by large veins, which swell up on the least vascular excitement or from slight compression of the neck. The eyes are not particularly prominent. The surface of the head is uneven from small exostoses and the thickening seems to be universal.

CASE IV. is that of a woman, thirty-five years old. The enlargement of the head was first noticed twelve years ago or more, and is now greater than in any of the other cases, though the symptoms of discomfort are less marked. In fact, the only symptom of note has been a tendency to severe burning sensations on the right side of the head and face, with a sense of numbness in the face, recurring in attacks of a few moments duration. Besides this she has a feeling of debility and a sense of fullness in the head. The enlargement is universal, but, as in the last case, it is of the cranial type, not affecting the face below the forehead, and not causing noticeable prominence of the eyes, though the edge of the orbits are thickened and rounded. The surface of the head is very irregular and is covered with bony

prominences of different shapes. There is a thick growth of coarse gray hair, but otherwise the nutrition of the skin and its appendages seems unchanged. In none of these cases was anything observed analogous to the general trophic changes seen in Dr. Starr's patient, except that in the first case the skin was of a yellowish color; in the second, that the neck was rather thick, and in the third, the hair coarse.

#### DISCUSSION.

Dr. STARR, of New York.—I have nothing to say, Mr. President, excepting that these cases are more advanced and more extreme than the case I reported in the *JOURNAL* to which I believe Dr. Putnam has referred. The heads are much larger. I think that the pathology of it is perfectly well shown by these sections of the skull. The origin of it I do not suppose any of us know anything about. And as for treatment I don't suppose any one knows any more about that. I tried thyroid extract with my patient because I had some rather remarkable and interesting results in acromegaly by giving thyroid purely as a tentative measure, but in my patient the thyroid treatment did not do any good whatever. My patient died of pneumonia, and I did not secure an autopsy.

Dr. PRINCE, of Boston.—Apropos of the last remark of Dr. Putnam I will briefly narrate a case, an account of which I have already written for Dr. Putnam, but which I have not with me. In this case the enlargement was not as general as in Dr. Putnam's cases. About three years before I saw this patient he met with a severe accident, viz., he was hit on the forehead by some tackle, and the story was that he had not been quite himself since then. At the time I saw him he suffered from symptoms of brain pressure very similar to those of cerebral tumor,—nausea, vomiting, headache, somnolence at times, loss of memory, and more or less of dementia. I saw him in consultation with a view to operation. In his case there was a large protuberance largely confined to the frontal bone. It was evident that the parietal bones were enlarged. Afterwards I was sorry that I decided against operation. I did not know how extensive the ostosis might be, and it seemed as if the operation would be a difficult one. Afterwards he died. There was no paralysis of any kind when I saw



him, but at the time of his death I was told by his wife that besides a certain amount of dementia and loss of memory, paresis of the left side of the body had been present. When we made the autopsy we found the thickening was almost entirely in the frontal bone. The orbital plates were enormously enlarged, so that they sprang up well into the cavity of the skull and left a very small space between them. The brain was squeezed downwards into this space between the orbital plates. The convolutions were flattened and smaller than normal. It was evident that the brain was compressed, pressed upward and downward. I thought from the examination of the skull that it would have been feasible to have relieved the condition by operation, and in such cases as this I should say an operation could be performed. That certainly was the impression of all who saw the autopsy, though the case was probably an exceptional one. There was no history of syphilis in this case, if I remember rightly. There was marked exophthalmos, but the man said his eyes always had been prominent.

Dr. HAMMOND, of New York.—I should like to ask if Dr. Putnam has seen in his cases evidences of structural changes in the skeletal apparatus elsewhere in the body. In a case I have seen recently almost the counterpart of the one presented here, there were five or six of these enlargements, and the skull was fissured and cracked in all directions. He was a syphilitic case, and four years previously, while driving, his horse stumbled,—he was driving with one hand,—he jerked the reins and broke his humerus; and about a year after that in pulling a cork out of a bottle, he broke his clavicle. The bones showed wherever they could be felt, enlargements and nodosities, and it is quite probable that in many of these cases the same cause that produces the structural changes in the bones of the skull does it elsewhere in the skeleton.

Dr. PUTNAM, of Boston.—That is one of the interesting points in connection with this matter, and I presume where syphilis is connected with the case the latter should be in a different class from those where no such cause is present. In a certain proportion of cases there does seem to be a tendency to enlargement of the bones through the rest of the body and changes of nutrition of various sorts. Virchow alludes to that fact, although the case on which he relied has been made out to be a

case of acromegaly probably. In Dr. Starr's case enlargement of the vertebræ was noticed. There is a tendency to bony enlargement. That would seem to militate against the view that the disease is distinctly due to spread of inflammation from some special point of injury. Possibly some toxic process goes on which affects the osseous system at large. Virchow, although he thinks the disease is inflammatory, speaks of predisposition in certain persons, and he means predisposition to include the tendencies related to certain epochs of development and other influences of that order, and that would include a good deal of what we now mean under the head of dystrophic tendency.

Dr. MILES, of Baltimore, showed

#### A CAST OF A BRAIN AND A BRAIN PRESERVED IN ALCOHOL.

Dr. WORCESTER, of Danvers, Mass.—During my service at the Arkansas State Lunatic Asylum I had the opportunity of making autopsies on three patients, in whom there was a very decided asymetry in the two hemispheres. In one case one hemisphere weighed seventeen ounces and the other nine. I will not say there was asymetry of the cerebellum in those cases. I examined the cerebellum in each of those cases and there was nothing that attracted my attention as to a difference in size between the different hemispheres of the cerebellum, although I might have overlooked a slight difference, I do not think I should have been likely to overlook any very striking one.

Dr. MILLS, of Philadelphia.—Many years ago I exhibited to the Philadelphia Pathological Society the brain of a negro murderer who had been confined in prison in that city for some time. In this case one of the most marked peculiarities of the brain was the atrophy of one cerebellar hemisphere co-incident with atrophy of the opposite cerebrum and particularly in the motor region. In a limited area of the pons was an old hæmorrhagic cyst, apparently the only active lesion which had been present in the case in this patient, and it seemed to me that this cyst in this particular locality had determined the atrophy both of the cerebellum and of the cerebrum.

Dr. STARR, of New York.—I regret that Dr. Peterson is not here to add to this discussion. Dr. Peterson has a

very large service at Randall's Island in the Children's Hospital for Idiots and has a number of these brains. I am indebted to him for photographs which I show to my students, of atrophy in various parts of the brain, and they are usually, I think, uniform in this, that you find an atrophy of the cerebellum corresponding to an atrophy of the opposite cerebral hemisphere, and you may depend upon it that the condition is congenital or acquired before the fifth year. Secondly, Dr. Peterson has some beautiful specimens of atrophies in different localities, and it is interesting to find that in these congenital cases the atrophy of the cerebellum accompanies only atrophy of the frontal or motor zone. If there is atrophy of the temporal or occipital region congenitally, we have no atrophy of the opposite half of the cerebellum.

Dr. BOOTH, of New York.—I have a case in point which I reported two years ago. In the brain an acquired cyst was developed after infection from scarlet fever at the age of five, and the post-mortem was held at about the age of thirty-three. There was a diminution in the left cerebral hemisphere and in the right cerebellar hemisphere. And the diminution either in the cerebral or cerebellar hemispheres was in the same proportion, not simply difference in weight, but the proportion of loss was the same.

## Asylum Notes.

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*The seemingly easy yet oft repeated problem* of the disposition of the chronic insane, whether they shall be by themselves or mingled with acute cases, comes up again as still unsettled in connection with the new Asylum at Wernersville, Pa. Dr. S. Weir Mitchell, in his oft quoted address, referred very favorably to this asylum; though others could not see there much more than was routine and customary in the chronic portions of our large country asylums. Several objections to this method are outlined in the report of Dr. H. D. Meredith, Superintendent at Danville, Pa. (1894). We quote as follows:—

“In July, 1894, fifty male and thirty female chronic cases were transferred to the same institution. While the act creating this asylum stipulates “chronic cases, —those who have been insane for one year or more,” as eligible for admission, and those sent included none who were helpless or dangerously inclined, ten men and twenty-one women were subsequently returned as undesirable, making the relief from this source sixty men and nine women.

Further relief is promised in the near future, but as it contemplates only quiet, cleanly and able-bodied cases, such as are capable of doing a fair day's work—and precludes those who are subject to periods of disturbance, the destructive, uncleanly, epileptic, and partly helpless—will merely reduce the total number resident without giving that relief hoped for and so badly needed. A forced removal of this class, while almost depopulating the better wards of the Institution, leaves the already overcrowded ones devoted to the care of the more disturbed, the demented and physically feeble, in the same condition, as an equitable division of numbers would render a proper classification impossible. Moreover, the removal of this class deprives us of that assistance from patients, upon which the institution has so long and successfully depended for an economical administration, and will increase the per capita cost to the full limit of \$3.75 per week, and may diminish the

usefulness of the Institution because of the inability to exceed this statutory limit.

In the establishment of a system of separate care for the chronic insane, it was advanced as a special advantage to the Institution having the restoration of acute cases as their main object, that the removal of the disturbing element which association with chronic cases was supposed to produce, should increase their usefulness, thus making them Hospitals in a truer sense. The removal of the quiet, inoffensive class, who could have no deleterious influence, even should they associate intimately with recent cases, and compelling their association with disturbed and violent cases, the very helpless and uncleanly, perhaps witnesses the distressing exhibition of epileptic seizures, and subjected to the annoyance of its attending irritability, is a travesty upon reason, and defeats the prime object of a chronic asylum as above set forth."

**Dr. C. Eugene Riggs** read before the Ramsey County Medical Society of Minnesota a plea for a state colony for epileptics. As he had recently come from a study of the Bielefeld colony of Westphalia, his account of this pioneer and most successful colony was very interesting. It is described as a village of 3000 people, located in a valley, and on the slopes of the hills surrounding it. Not all of the 3000 cases are epileptics.

The work seems to be done by a society of deacons and deaconesses, who act as teachers, friends, nurses and attendants. Many trades are provided to meet the accustomed work of each patient. There seems to be given but little and ordinarily no wages. Some of the occupations pay for themselves and with a profit, others do not. Bromide is mentioned as seemingly the one drug used to combat the disease. The institution is supported as a whole by voluntary contributions.

The colony near London, the one in Ohio, and the one in California, as well as the one just planned in New York State, are mentioned with some detail. An estimate is made of from one to two epileptics to each 1000 people; and the need of this so large a class is detailed as calling very strongly upon the State to make a light provision for the segregation, the home-like and careful care, the protection of themselves and the protection of others from them, in this and other States.

*In the Medico Legal Journal* for March, are to be

found more opinions from alienists concerning the conduct of Hospitals for the insane, and is a continuance of the list previously noted in this Journal (Feb. '95). Sixteen more letters are included. The most essential question, should the physician be excluded from financial or business matters, is answered variously as before. Of the forty-three writers in all here collected, thirty-two wish the Superintendent's power to be complete, although nine of these disclaim any wish except for a veto or Supervisory power. About eleven are in favor of a complete separation.

A summary of the arguments used in this and recent literature may not be out of place, as this seems a question considered the most vital of any other debate. In favor of separation it is urged:—

1st.—One man has not *ability* to excel in both business and medicine.

Moreover, physicians are not apt to have had business training, even if they have economic business sense.

2nd.—One man cannot possibly find *time* to excel both as a business man and as a scientific man.

3rd.—Admitting that it is often voluntary with Superintendents, whether they spend the majority of their time among the business interests or among the medical and scientific matters, it is yet claimed that the demands and tendencies all carry toward the business matters unless such is prohibited. Especially as, unavoidably and permanently a more or less political element enters into nearly all offices in the State.

It is hard to avoid such pre-occupation, and the natural liking for power, given by the spending of large sums of money, fosters it.

4th.—The Superintendent being occupied with business, the Board appreciating best business success, the tax-payers and legislators pressing business ideas, the assistants are not so apt to be employed because of high attainment as because of inexperience or recent graduation or willingness to accept small salaries temporarily. History confirms this view, though exceptions, of course, are many.

5th.—Excluding the engaging of nurses and custodial help, the ability to regulate diet and ward furnishings and control of the kind and amount of patients' labor, it is claimed that the multitude of other businesses do not effect the patient's life or comfort, any

more than nor *as much* as they would sane people in surgical or general Hospitals. Though stated otherwise, it is not yet proven.

The arguments on the other side may be summed up about as follows:—

1st.—The business head or manager would clash or interfere with a medical head. The presumption here is that the clashing referred to is the business interest, interfering with medical aims. It has been stated that two heads are as absurd as two heads on a single body.

2nd.—The business affairs have a medical relation: it is held that even outside works, farming, engineering, gardening, slaughter-house, soap-house, carpentering, etc., have medical relations which need the decisions of a medical head.

3rd:—Many alienists take a nominally modified standing, asking only for a consulting power upon prominent business topics, or for a veto power, or for a supervisory power. (Indeed this is nominally the position of many, while their actual duties vary quite widely.)

4th:—Finally it is to be noted that a modified plan is being suggested. A physician in general charge of the executive work and another physician in general charge of clinical and pathological work. This might be called a compromise, in that it secures the so-called desirable decision of farm, building and supply questions by a medical man. If a man in charge of the clinical and pathological work is unhampered and receives due honor, it would seem quite practical.

***The "section of nervous and mental diseases"***

at the Minnesota State Medical Society, which met at Duluth June 19th-21st, inclusive, eventually resulted in four papers exclusively upon the subject of insanity, probably the first occurrence of the kind in the history of the State. Nor were the papers formal or routine in any way, but were such as were calculated to incite new and revived thoughts along the line.

The papers presented were "The Ideal Hospital for the Insane" by Dr. R. M. Phelps, "Modern Treatment of the Insane" by C. Eugene Riggs, "The Clinical Study of the Individual Insane," by H. A. Tomlinson, "The Development of the Insane Asylum," by Dr. Helen Bissell.

The papers were actively discussed and a practical outcome was reached in the form of a resolution that the next hospital built, now provided for by law, ought to be

located near the two large twin cities, to be available for study and instruction and to be also more logically and economically near its supply of patients.

*The Transactions of the American Medico-Psychological Association* for 1894 are but recently out. They are presented in a bound volume and in very acceptable shape for preservation. The articles have long since been presented in the various Journals, but this grouping of them is a very useful thing for alienists. It is published in Utica, N. Y., by the press of the Utica State Hospital.

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*A contribution to the knowledge of the course of the fillet superior, cortical or thalamus-fillet.*—Jacob (*Neurolog. Centralblatt*, 1895, No. 7).

A report of two cases, in both of which there was an interruption of fillet system by a focus. In the first case an extended focus had totally destroyed the thalamus, the subthalamic region and the tegmentum of the posterior corpus bigeminum of one side, the fillet was completely interrupted in this region. The result was a total disappearance of the fillet down to the crossed nuclei of the posterior column, which condition must be considered as descending degeneration.

In the second case there was a total destruction of the cortex and medullary stratum of the left hemisphere, including also the upper part of the internal capsule and the whole putamen. Everything else uninjured. Here the left fillet was somewhat narrower than the right but not degenerated; simple atrophy of moderate degree.

These findings confirm Mahaim's view that the fillet becomes interrupted in the subthalamic region, while they prove the error of Hoesel's statement of the uninterrupted course of the fillet fibres from the nuclei of the posterior columns in the cerebral cortex.

Although cases of ascending degeneration of the fillet have been reported the author believes that most of the neuron cells giving origin to the fibres of the fillet are situated not in the nuclei of the posterior columns but in the subthalamic region (perhaps basal parts of the thalamus or globus pallidus). Here they may become connected with the terminal arborizations of a second neuron which has its cell centres in the cortex. Or, the other possibility is that homologous to the peripheral sensory neuron which has its cell centre in the spinal ganglion, the whole course of the fillet system, from the nucleus of the posterior column to the cerebral cortex of the opposite side, is supplied by one neuron having its cell centre in the subthalamic region. Such an explanation, in favor of which the author intends to produce arguments in a future publication, might reconcile the contradictory findings and views of Hoesel and Mahaim.

ONUF.



## Periscope.

### CLINICAL.

**Congress of German Surgeons.** *Medical Week*, April 20, 1895). Of the many important and to neurologists, extremely interesting contributions made before the German Congress of Surgery the Address of Von Bergmann on "Recent Progress in Cerebral Surgery," and the Report by Kocher on "Six Thousand Extirpations of Goutre," merit the closest attention. The former said that, thanks to the introduction of antiseptic methods and the steady advance realized by physiologists and clinicians in the domain of cerebral localization, surgeons now unhesitatingly, and frequently with success, undertake the treatment of diseases which have hitherto been regarded as incurable. Unfortunately, for various reasons, and particularly the difficulty or impossibility of arriving at a correct diagnosis in many of the cases, the field of cerebral surgery will always be limited. Thus, with regard to tumors of the brain, statistics show that operation is practicable only in 29 per cent of all cases, and that three-fourths of these cannot be operated upon on account of the impossibility of arriving at a diagnosis. All our efforts should, therefore, be directed, on one hand, toward defining the diagnostic indications, and on the other hand, toward perfecting more and more our operative methods. Considerable advance has already been made in the latter direction by the introduction of extensive osteoplastic resections of the skull, which render possible the extirpation of large tumors. Nothing is of more importance than speed in operation, and this can be obtained by the use of an instrument which I am enabled to show you, consisting of a circular saw, set in extremely rapid motion by means of a small electric motor. With this instrument a large bone flap may be sawed out in a few moments, the operation being completed by the aid of a sharp chisel, in order to avoid, as far as possible, injuring the dura-mater.

Numerous attempts have been made to cure epilepsy by surgical treatment; but these attempts have proved unsuccessful, except in those forms of epilepsy which are due to a lesion of the cortical motor centres. It is necessary, however, to be guarded in drawing conclusions from the results obtained, as cases of temporary recovery of epileptic patients, consequent upon simple trephining or excision of a cicatrix of the scalp, are of frequent occurrence. For my own part, I have seen epileptic attacks cease during the time necessary for the healing of the wound in an individual who had undergone amputation of the leg. It is impossible to approve of operations, which consist in extirpating, in epileptic patients, portions of the cerebral cortex which are apparently healthy, but which are considered to be the starting-point of epileptic seizures. Extirpation, indeed, is only justified in cases in which a distinct lesion, a cyst for example, exists.

Great success has been obtained in operations for abscesses of the brain. Surgeons have even had the courage to attack thrombosis of the sinuses and suppurating leptomeningitis. In this case also the diagnosis is far from being always possible, and in many instances of unsuspected abscess intervention is, therefore, abstained from. In the very short time at my disposal I cannot deal as fully with the treatment of intracranial abscesses, as would be desirable in view of the importance

of this subject. I shall, therefore, confine myself to briefly considering the most frequent form of these abscesses, viz., those which follow a suppurating otitis media. In one-fourth of such cases, the cause of the suppuration is a cholesteatoma of the middle ear. Intracranial abscesses, developing under such conditions by continuity, are situated either outside, or within, the dura-mater, or in the interior of the cerebral substance, most frequently in the temporal lobe. In order to reach these abscesses, which are comparatively accessible and, as a rule, located in the portion of the cranial cavity situated above the tympanum, I have devised a method of operation, which, in my opinion, is preferable to the simple trephining usually resorted to in such cases. This procedure consists in resecting a quadrangular portion of the wall of the skull immediately above the external auditory meatus. With this object in view, a cutaneo-musculo-periosteal flap, left adherent at its upper part, is made by three incisions—one horizontal, beginning above the zygoma and ending behind the mastoid process, the others vertical and perpendicular to the former, one in front of the tragus, the other behind the mastoid process, both being carried upward to a height of about 4 centimetres. Removal of the bone beneath this flap gives unobstructed access to the interior of the skull, and greatly facilitates the task of the operator.

In thrombosis of the sinuses, surgical intervention has given unexpected results. The object of this intervention, as is well known, is to reach directly the transverse sinus—which is the one usually involved—through the mastoid process, the transverse sinus being situated beneath the middle portion of the latter. It is unnecessary to insist upon the details of this operation; incision of the sinus after exploratory puncture, evacuation of the clots, disinfection and plugging, and lastly, ligation of the internal jugular vein. Of thirteen cases operated upon, recovery followed in six. Death is usually due either to pyæmia or to suppurating leptomeningitis.

Of late, operations have been undertaken in order to remedy the inconveniences resulting from exaggerated intracranial pressure, from whatever cause. In this connection Quincke's attempts at tapping the lumbar portion of the spinal cord, in cases of basilar meningitis, are worthy of mention. The results obtained so far have only amounted to some improvement of short duration. In no instance has a fatal termination been averted by such puncture, but, on the other hand, it has proved useful as a diagnostic measure, because, if bacteriological examination of the liquid withdrawn reveals the presence of Koch's bacillus, it is evident that it is a case of tubercular meningitis. Possibly puncture may also be of value as an aid to diagnosis, when the existing symptoms do not permit of a differential diagnosis between abscess of the brain and more or less localized suppurating meningitis.

In commenting on his "Report of Extirpation of Goitre," Kocher remarked during the past ten years in which he had performed the operation on 900 patients, he had met but one case in which cachexia strumipriva had developed. This was entirely owing to the fact that he always left a part of the thyroid gland, which was sufficient to carry on the functions of that organ. In the single case in which the cachexia developed the extirpation was unilateral, but after the operation it was found that the other side was atrophied.

A very interesting statement by Kocher is that he had several times noticed that tetany, which he is pleased to call acute cachexia, developed. It is interesting, likewise, to hear that the patient in whom cachexia strumipriva developed recovered through thyroid feeding.

In speaking of the mortality in his operations, Kocher says that he deducts thirty cases of malignant goitres in which unusual and peculiar difficulties militated against success. Of the remaining 870, eleven died; but in six only was death the direct result of the operation, and of these

three were operated on for Graves' disease. The extirpation of the goitre in the latter disease he regards as dangerous. For the latter he prefers to ligature the thyroid arteries; but never more than three of them.

Referring to some researches that had been made under his observation by Lanz and Trachewski in the treatment of goitre by the ingestion of thyroid extract, the author said that in the long run this method may determine complete atrophy of healthy parts of the thyroid gland. He further remarked that all the symptoms of Graves' disease had been produced in healthy animals by these experiments. He found also that the symptoms of exophthalmic goitre improved greatly under treatment by phosphate of sodium.

J. C.

**Intracranial Hæmatoma with Collateral Paralysis.**—Ledderhose reported a case of intracranial hæmorrhage with collateral paralysis, that is to say, paralysis affecting the same side as the extravasation. The patient was a man, aged thirty-eight, who was knocked down in a fight and kicked on the head. Though he lost consciousness for the moment, he was able in a short time to walk home, but during the next few days he gradually developed symptoms of cerebral depression. At the end of nine days he was obliged to take to his bed. When seen three days later he was in a comatose state, with paralysis of the entire right side of the body. A diagnosis of intracranial hæmorrhage from laceration of the left middle meningeal artery was made, although there was no sign of a fracture. No hæmorrhagic focus was found. The patient died on the day following the operation. At the autopsy it was proved that there was neither fracture of the skull, nor laceration of the middle meningeal arteries; but on the right side, corresponding to the paralysis, there was within the dura-mater a large hæmatoma, covering the middle and posterior portion of the corresponding hemisphere. On carefully scrutinizing the literature of the subject only thirteen cases of collateral paralysis, consequent either upon cranial traumatism or other pathological lesions can be found. Brown-Séquard was the first to call attention to this condition. On the other hand, researches by various investigators, Pierret among the rest, have shown that abnormalities and individual differences frequently exist in the manner in which the fibres of the cerebral hemispheres cross each other. It may even happen that they do not cross at all, and this fact explains the possibility of collateral paralysis. Cases of this kind may be a source of great embarrassment to the operator, as it is usually impossible to diagnose the site of a lesion on the same side as the paralysis. Sometimes, however, the existence of an ædematous swelling of the optic disc on the paralyzed side suggests such an abnormality. Be this as it may, when in a case of this kind, trephining does not enable us to find an extravasation where it was expected, one is justified in making a fresh opening in the bone on the side corresponding to the paralysis.

J. C.

**A case of lead-poisoning, with extremely rare symptoms.**  
—Tankowski (*Neurologisches Centralblatt*, 1895, No. 7).

The peculiarities of the case were:

1st. The unusual (? Ref.) manner of poisoning. The patient was a plumber and had to solder water-pipes with a mixture of lead-peroxide ( $\text{Pb}_3\text{O}_4$ ) and shellack. In the author's opinion the poison could only have entered the body per os, the patient touching his food with unclean hands, thus introducing the poison with which they were soiled into the digestive tracts. All other avenues for entrance of the lead could be excluded.

2d. The pulse which was hard at the time of the admission to the hospital, became filiform and irregular twenty-four hours afterwards, marked embryocardia, without apparent reason, no organic lesion of the heart walls or valves being present. Tankowski has not found

such a condition of the pulse mentioned as in constant connection with lead poisoning, yet in his case no other cause but the lead poisoning could have produced it.

3d. There was a paresis of the lower branches of the right facial nerve. Aside from general statements in text-books on nervous diseases, Tankowski has not met with any cases of lead poisoning in the literature that were accompanied by said symptoms.

4th. The right pupil was smaller than the left and showed diminished response to light. Only one similar case has been reported (by Déjerine-Klumpke).

That the case was one of lead poisoning was proven by the presence of lead, the gums which showed a grayish-blue line on which two reactions were made. A piece of absorbent cotton drenched with peroxide of hydrogen was pressed against the gums for several minutes, when it was found that the gray line had changed into a white one (PC T—gray, insoluble—by oxydation becomes PC T O<sub>4</sub>—white, insoluble). The following second reaction was made: A piece of cotton was drenched with a solution of oxide of potassium to which a few drops of muriatic acid were added and pressed against the gums of the opposite jaw; when it was removed twelve minutes afterwards the gray line had turned yellow (by the mixture of H Cl with K O hydriodic acid formed which changed the PC T into PC T<sub>2</sub> an insoluble of yellow color). The presence of lead in the urine, proven by various reactions, was also proven.

ONUF.

**Tabes Arrested by Blindness.**—Déjerine (*Gazetta degli Ospet-ali e delle Cliniche*, 1895, No. 46).

Benedikt was the first who called attention to cases of tabes complicated with atrophy of the optic nerve, and who pointed out the peculiar fact that in these cases, with and by the appearance of said atrophy, the development of the tabes became arrested. He further stated that whatever stage the tabes had reached, the motor disturbances characteristic of tabes receded, as soon as the disease complicated itself with atrophy of the optic nerves.

Déjerine confirms the first statement, but contradicts the second. According to his experience, the atrophy of the optic nerves if it does complicate the disease, sets in at an early stage, viz, before the disturbances of motor co-ordination have made their appearance. He thinks that when the motor inco-ordination is once established the patient can be nearly assured that he will not become blind.

Mostly the atrophy appears at a time when the tabes is distinctly marked already by the accompanying lancinating violent lightning-like pains. After these have existed some time the vision then begins to diminish, not simultaneously in both eyes, but first in one, then in the other. It diminishes quite gradually, the visual field becomes irregularly narrower for the various colors, first for the green and red, then for the yellow, last for the blue, the final result being always complete blindness. Correspondingly, the ophthalmoscopic examination reveals, first, a grayish or gray-bluish papilla with sharp outlines, then a white papilla, and finally one that looks similar to mother of pearl with conservation of the calibre of the blood-vessels and without excavation.

From the side of the pupils there is to be noted inequality, myosis, mydriasis or normal dimension; but there is always absence of reaction to light and absence or diminishment of the reaction to pain, while the response to convergence remains preserved.

Simultaneously with the development of the blindness the lancinating pains nearly always diminish in intensity and frequency to disappear entirely in some cases.

In a second series of cases the amblyopia instead of following the lancinating pains at some more or less remote time, appears contemporaneously with them.

In the third series no pains exist, only the blindness is present. In these cases we probably have to deal with tabetics in whom the tabes has involved only the eyes, especially the optic nerves. The fact that in some such cases lancinating pains set in years afterwards (Gowers), and the pathological anatomical findings demonstrating the lesions of initial tabes prove clearly that we have before us a special clinical form of tabes.

The following facts are still noteworthy :

1. Sometimes the knee-jerks, after having been absent for years, appear again when blindness occurs.

2. The patients whose tabes has been arrested by the blindness do not present Romberg's symptom. They can stand steadily and walk as normally as a blind person can walk, showing no ataxy whatever in their gait.

3. The prognosis of the optic atrophy is fatal. Two cases illustrating the author's deductions are reported. In one case the lancinating pains disappeared after blindness had set in, while the gastric crisis remained uninfluenced.

ONUF.

#### PSYCHIATRY.

**A Case of Periodic Paranoia.**—Ushenko (*Archiv's. for Psychiatry, Neurology and Legal Medicine*, Russian, 1894, Vol. iii.)

The article begins with a review of the casuistic of the disease which shows that only three cases have been reported so far, one by Dagonet, (doubtful case), the second by Mendel, the third by Kausche. The author then describes his case: Family history.—Father a crank and an alcoholic; one brother a crank, women hater; has only four fingers on one hand; another brother has a marked curvature of the spinal column. Patient's history.—Man of thirty-five; teacher; married; of somatic peculiarities and irregular development of the teeth; frequent masturbation during five years, occasionally practiced such during his married life.

The first symptoms of mental disease were noticed in the spring of 1893, after an injury of the head, which was followed by erysipelas, although it is probable that mental aberration began earlier, and at first passed unnoticed. The development was gradual. The patient at first unsocial and distrustful became suspicious; watched keenly the people about him and saw in everything sinister intention towards himself. This distrust gradually developed into typical delusion of persecution with all its characteristic features. Later delusions of greatness also developed. Gustatory and visual hallucinations and occasional hallucinations of hearing somewhat tend to confirm him more in his delusory ideas, but do not play an important part. This condition which bears all the characteristics of paranoia (primäre Verrücktheit, délire systématique, folie systématisée) alternates with periods of perfect mental lucidity.

The patient was admitted to the asylum in November, 1893, where he presented the following condition: Complete orientation inasmuch as he knows that he is in the Charkow Asylum, as he recognizes the physicians and acquaintances visiting him, etc. But he pretends that the actual designation of the asylum is to prepare its inhabitants (which he says are no patients) for prominent positions. He believes that another patient by means of electricity and magnetism knows all his thoughts and smiles at them. Absorbed in his thoughts he pays scarcely any attention to happenings in his surroundings, so that frequently the most common things escape his notice. But sometimes meaningless things excite his attention to a high degree; for instance, he anxiously and suspiciously watches how the patient formerly mentioned rubs his hands, which he considers as means to magnetize him. He imagines himself to

be an extraordinary person, the people expect great deeds from him, perhaps he will make great reforms or become emperor, authors reciting of services have him in view, etc. He has illusions of sight, inasmuch as he sees peculiar and actually not existing expressions, smiles, etc., in the face of his surroundings. Sometimes he tastes poison in the meals. He heard somebody call "anti-christ." Another time he heard the exclamation: "What is form." Whether he had more hallucinations it is hard to tell, but evidently they did not play a prominent part in his psychical life.

During his stay in the asylum the patient had an attack which begun before his admission to the asylum and lasted six days. Then he had an interval of perfect lucidity, lasting about one week, which was followed by a second attack of about two weeks' duration.

The characteristics of the case are: 1. The alternation of the attacks with intervals of lucidity. 2. The resemblance of the attacks between each other. 3. There is only a short premonitory period to the attack. 4. The whole personality becomes changed at the time of the disease. 5. Full understanding of his disease during the lucid intervals.

The author comes to the conclusion that periodic paranoia belongs to the psychoses of the degenerated, and characteristic of these psychoses is that no matter how frequently they may occur in an individual, they seldom lead to dementia.

ONUF.

**Contribution to the Doctrine of the Infectious Origin of Acute Delirium.**—Branchi and Piccino (*Annali di Neurologia*, 1894, fasc. vi).

The result of this investigation of the authors confirms their views of the infectious origin of delirium acutum, as published in a previous article (*Annali di Neurologia*, 1893). In the present monograph they report eight cases of hallucinatory delirium. In seven cases recovery or considerable improvement took place; the eighth ended fatally. In the eighth case only did an examination of the blood reveal the presence of the bacilli which the authors had described in their previous article and which they consider to be characteristic of the delirium bacillare (delirium grave). In all the other cases the blood contained micro-occi arranged in chains or grape-like, isolated or in pairs, staining with the usual aniline stains. Among the micro-organisms isolated from the blood, the authors noticed the streptococcus pyogenus (in four cases) and the staphylococcus aureus (in two cases).

The comparison of the findings in all cases mentioned leads the authors to the following conclusions:

1. Among the forms of mental disease which clinically bear the aspect of delirium acutum and have been described as such so far, there is one which might best be designated as "delirium acutum bacillare." This form differs from all others clinically by the greater intensity of the symptoms, by the adynamic stage which directly follows the stage of excitation, by the short course, and by exitus letalis; bacteriologically by the presence of a special bacillus in the blood and in the nerve centres.

2. Fever is not characteristic of the bacillary nature of delirium acutum; there are other febrile forms of acute sensory delirium in which no other cause (pneumonia, bronchitis etc.) can be attributed to the origin of the fever except the same which produced the delirium.

3. The delirium acutum bacillare must be considered as a grave infectious disease in which the symptoms of excitement and of grave disturbances of consciousness are succeeded by symptoms of depression and a grave typhoid-like condition.

4. In the other forms of acute delirium and mania gravis the bacteriological examination gives also positive findings, but other than in the delirium acutum bacillare. In these other forms grave disturbances of nutrition are observed, accompanied by fever and other toxic effects on the functions of the central nervous system, the greatest disturbance of which consists in the hallucinatory delirium.

That Martinotti and Abundo did not find the bacillus mentioned, but other forms of micro-organisms in the cases of sensory delirium which they investigated, proves first that the forms of delirium observed by them were not the delirium acutum verum (bacillare); second, that these other forms of sensory delirium and mania gravis are much more frequent than the delirium acutum bacillare, which is a very rare disease.

5. The organic stupor must be considered as a secondary stage of the hallucinatory delirium, not only because exact observation always demonstrates the pre-existence of a hallucinatory stage, but also by the identity of the bacteriological findings in the hallucinatory stage and that of stupor.

ONUF.

#### ANATOMICAL.

**The Staining of the Nervous System.**—Flatau (*Deutsche Med. Wochenschr.*, No. 13, 1895).

During a demonstration of specimens of the nervous system, Flatau said, speaking retrospectively, that in 1865, Deiters proved the existence, in every ganglionic cell of the nerve centres, of two kinds of expansions: some, branching, which have been described as protoplasmic processes, and another, finer filament, which has become known by the name of axis-cylinder. Almost at the same time, Gerlach showed that the protoplasmic processes were united to each other, forming a network which was known as Gerlach's reticulum. In 1880, Golgi proved that the reticulum does not exist, and that the protoplasmic processes terminate in free extremities. Golgi, however, assumed the existence of a reticulum formed by anastomoses of the collateral and terminal branchlets of the axis-cylinders; but, in 1889, Ramón y Cajal proved that this reticulum does not exist either, and that the ends of the prolongations of the axis-cylinders in the nervous system are entirely free.

To the nerve cells, as thus understood, that is to say, completely independent unities, which never anastomose either by their protoplasmic branches or axis-cylinder expansions, Waldeyer gave the name of neuron. The terminal arborizations of the axis-cylinder prolongations and the protoplasmic prolongations, which may be compared to the branchings of a bush, have come to be known as "end branches." Waldeyer has recently, in a work not yet published, decided to substitute the word "Polstücke" instead of "Endbäumchen."

The theory of the neuron simplifies greatly our conception of the structure of the central nervous system. Heretofore, for instance, it was supposed that the retina was composed of a large number of layers, while to-day it is generally admitted that it consists only of three neurones: the sight cell, the bipolar cell, and the ganglionic cell.

Is it possible to explain the physiological functions by the theory of neurons? Let us take motion, for example. A neuron, the motor cell of the pyramid, connects the terminal expansion of its axis-cylinder with the protoplasmic processes of a motor cell of the spinal cord, with the result that the impulse is transmitted to the nervous ramifications of the muscles and determines contraction. According to this theory, motion is the result of the transmission of a nervous current by two neurons, one of which is placed at the starting-point (archineurone), and the other (teleneurone) at the termination of the process.

The sensory phenomena are also transmitted by two neurons, but in this case the cell of the spinal cord acts as archineurone, and the cell of the cortical layer as teleneurone.

In certain cases there may be an intermediary neurone.

All reflex acts may also be explained by conduction from neurone to neurone.

With regard to psychical phenomena, it is probable that they are the result of bringing into contact a greater or less number of protoplasmic cell expansions.

One can say with certainty that the neuron is not alone an anatomical, but a physiological entity. The important question which remains to decide is, whether the neuron is a functionary entity. To answer this question one must enter the domain of pathology.

The body of the cell plays the principal part in the life of the neuron. When it is changed, its prolongations degenerate. The death of the cell invariably precedes that of its prolongations.

In the sensory region the application of the neuron theory is most fitting to our conception of tabes.

According to Charcot and his followers, the degeneration of the posterior columns in tabes is a primary lesion. Leyden, on the other hand, looks upon the degeneration of these columns as always preceded by degeneration of the posterior roots.

Wollenberg, in a case of tabes, found that the cells of the spinal ganglia were diseased, although he considered the lesion too slight to have been the starting-point for the tabetic process. Fresh investigations by Erb and Strümpell, however, showed that the motor cells exert a paramount influence on the peripheral portion of the nerve fibre. We are constrained to assume that changes take place in the spinal cells, which cannot be discovered under the microscope, but are, nevertheless, sufficient to determine degeneration of the posterior columns. The lesions discovered by Wollenberg in these cells are, therefore, in all probability sufficient to explain the degeneration of the posterior columns. J. C.

**Experimental Investigation of the Changes Found in the Ganglionic Cells, in Acute Alcoholic Poisoning.**—Dehio (*Centralbl. f. Nervenhk. u. Psych.*, March, 1895), experimenting on animals, in order to study the structural changes of the central nervous system, opens a new field for psychiatric investigations; Rabbits and dogs were poisoned with alcohol, the autopsy was made immediately after death, and the central nervous system examined microscopically. The changes in the ganglionic cells of the cortex were studied carefully, especially the Purkinje's cells of the cerebellum. By the aid of Nissl's methylene-blue staining method, the lesions of the nerve cells could be detected with great exactitude. The animals that died in a few hours (quick poisoning) showed such slight changes, that they could not be referred to alcoholic poisoning with certainty. The cases, in which the intoxications lasted for some time, were characterized by the following pathological lesions in the Purkinje's cells: 1. The tingible substance showed, instead of the delicate network, fine granula of equal size, which were distributed irregularly. 2. The non-tingible substance had taken a light blue shade. 3. The changes appeared in the entire cell at times, at others only in certain parts. The fine rows of granula in the prolongations remained unchanged. 4. The nucleus and nucleolus remained unchanged (with the methylene-blue stain). 5. All the ganglionic cells were by no means impaired, even where the changes were most advanced; in many parts there were whole rows of normal cells, and between them the pathological. The author is quite conscious of the fact, that the foregoing observations cannot explain the pathological effect of alcohol to any extent; but the positive result obtained teaches that experimentation on animals may be made use of in psychiatry and psychology, and that pathological lesions may be found in disturbances hitherto considered functional. MACALESTER.



## Book Reviews.

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E. BRISSAUD. LEÇONS SUR LES MALADIES NERVEUSES, (*Salpêtrière*, 1893-1894), Recueillies et Publiées par Henry Meige. Paris, 1895.

The appearance of a new work on diseases of the nervous system from the clinic of the Salpêtrière will probably arouse considerable interest, not unmingled with curiosity, now that Charcot is dead. The medical world was so accustomed to greet with favor the productions of the great French neurologist, and to expect an annual contribution from his busy pen, that it probably will miss sadly the gap in neurological literature which his lamented death has made inevitable. It can be said of Charcot, as it could be said of no other world-famed clinician, that his less was international; that the cessation of his marvelous clinical activity created the painful impression that not only the light of a great original teacher had gone out, but that even the vast fruitful field which he had in a sense created for the benefit of all nations, was henceforth to lie barren.

As is always the case this latter assumption was only the unconscious and unreasoning tribute which the world pays to genius. The man only departs: the fruits of his labor, his example and, above all, the opportunities that he created for others, remain and will long remain; they are in fact permanent. Of this we have a living proof in the book now before us; for it is, in a peculiar and laudable sense, a reflection of the great master, a continuation of his work in the midst of his own clinic, from out of which though being dead, he still seems to speak.

Brissaud has undertaken a delicate and difficult task, of which evidently he is painfully conscious, and in which he should have the sympathy and encouragement of all neurologists. In his modest preface he declares that his book is only a respectful homage to the memory of him whose name recurs in all its pages. He has evidently made an herculean effort, however, to produce a noteworthy book; one that will not only do honor to the memory of his predecessor, but will also redound to the credit of himself. In this he shows a sincere but shrewd appreciation of both his circumstances and his opportunities, for while he is mindful of the contrast in which he must stand with Charcot, he is not unmindful of the advantages which his temporary position in Charcot's clinic gives him before the scientific world. The result is really a noteworthy book, which if it is lacking in the individuality and lucidity of Charcot's style, equals if it does not surpass in illustrations and extrinsic embellishments even the best productions of that scientist. Brissaud has, indeed, used his materials and his opportunities for all that there is in them. In the rich field which he has inherited he has left nothing unappraised. Consequently the book presents a wealth of clinical material and a profusion of illustrations, mostly original, that are truly remarkable.

The general impression made upon us by the book is highly favorable. From the mechanical and artistic standpoint the volume is beautiful. Printed in large type on heavy paper it abounds in diagrams and photographs, many of which are of great merit. Most of these are original to the Salpêtrière, some of them we think we recognize as old acquaintances; while a few have been borrowed from other authors,

some of whom, it may be worthy of note, are not Frenchmen. In this respect, as also in his literary references, the author shows rather more disposition to do credit to foreign observers than is common in France. But in this matter he still has opportunity for improvement.

From the scientific standpoint the contents of the book are sound and meritorious. The treatment of each subject is conventional, and therefore rather commonplace. The author does not impress us as aiming to be individual and original; in fact, we rather think that he avoids any such pretence, except in rare instances, and is content to walk safely behind the shade of his great predecessor. In this perhaps he is wise; he may feel instinctively that in France it is too early for him to be aught else than the echo of a voice in Charcot's clinic.

This attitude is shown by Brissaud in his first lecture, which is on amyotrophic lateral sclerosis, or as he dutifully calls it, "Charcot's Disease." Such a nomenclature is always faulty, but it is, perhaps, excusable in this case. In his description of this affection he simply reiterates Charcot, and does not appear to take much account of the exceptions, based upon clinical observations, which others have taken to Charcot's original description. Gowers and Leyden, with reason, have noted that most of the cases formerly thus labelled were apparently only forms of progressive muscular atrophy; while Mott in a recent case, reported in *Brain*, found that the degeneration extended through the whole upper segment of the motor paths, including even the motor cortex. Whichever of these exceptions shall hold good—and both seem to be established—the fact will remain that our conception of this disease, as formed on Charcot's original description, must be materially modified.

One of the most valuable—and, by the way, original—chapters in the book is on Hæmatomyelia and Sudden Paraplegias. These apopleciform paraplegias have been too generally misunderstood, their importance underrated and the exact frequency of their occurrence ignored. Recently in America and England several important cases have been reported by Kindred, Starkey and others (but they are not quoted by Brissaud), which proved conclusively by autopsy that the symptoms were due to hæmorrhage within the substance of the cord. Formerly, largely due to the teachings of Charcot, Hayem and others, these hæmorrhages were thought to be secondary to a myelitis or an acute softening. The fact probably is that they are primary to the softening that is found in these cases, which is due to a necrosis caused by the hæmorrhage or by the cause of the hæmorrhage. The original cause of the hæmorrhage may be, as Brissaud points out, either trauma, or disease of the blood-vessels, by syphilis, gout, etc., or even emboli from a diseased heart. This chapter does something towards clearing up the obscurity that has hung so long about many cord-cases, which have been indiscriminately diagnosed as myelitis. It emphasizes the fact that diseases of the blood-vessels, which play such a conspicuous rôle in cerebral pathology, may have an equally malign influence in the pathogeny of diseases of the spinal cord.

Brissaud's lecture on *Tabes Dorsalis* is interesting, but it is devoted rather too exclusively to an attempt to prove that the arthropathies of this disease are the results of involvement of the sensory fibres. He makes of locomotor ataxia two types, as is the French mode, one purely motor and one purely sensory. The arthropathies occur only in the latter of these types. It is true that they occur in the mixed motor and sensory cases, which as we all know, are the usual types, but in these cases Brissaud concludes, rather dogmatically, that they depend upon involvement of the sensory structures. To support this thesis he refers to the arthropathies of syringomyelia and general paresis, claiming that in both these diseases the joint-changes are caused by disease of the posterior columns. He reproduces the photograph from an

American case of arthropathies in general paresis, in which a careful microscopic study had been made, and claims erroneously that the microscope shows systematic degeneration in the posterior columns as in locomotor ataxia; whereas the diagram presented with the report of the case, while it shows some degeneration in these columns in a rather limited area, does not show the typical lesion of locomotor ataxia, and does show extensive degeneration in one lateral tract. These, perhaps, are fine points, but they may as well be stated correctly. It seems rather arbitrary to attribute an effect to involvement of a few special fibres when other fibres are conspicuously diseased.

Brissaud makes an exhaustive study of the ophthalmoplegias in all their relations. This, in our judgment, is the most valuable contribution in the book, and, on the whole, is the best and most complete that we can recall at this moment in any book. It is well illustrated with photographs and diagrams, taken mostly from original cases. These well display the wealth of clinical material at the author's command, and are evidences also of the method and discipline of a thoroughly well organized clinic that make such a study and such a book possible. Brissaud first gives an admirable description of the nuclear anatomy of the mid-brain, and of the origins of the fibres of the third and fourth nerves, as well as of the sixth, and their mutual relations, and their relations with those of the opposite side. The demonstration is largely diagrammatic, as it is bound to be in any man's hands for the purpose of teaching, and the author falls back upon the rather nebulous theory of *functional* centres as distinct from *anatomical* centres, in order to explain the complex coördination of the extrinsic and intrinsic muscles of the eyes. He treats the subject clinically in its relations with the orbit, the sub-peduncular region, the intra-peduncular region and the mid-brain nuclei; and, moreover, gives details of a case of ophthalmoplegia, of the type described by Hutchinson, in a woman affected with progressive muscular atrophy of the four limbs. This last observation is one of exceeding interest. The author relates and illustrates a second case, also in a woman, of partial ophthalmoplegia with hemiatrophy of the tongue, showing the effects of a combined superior and inferior polio-encephalitis.

But why, let us ask, should the crossed paralysis of the third nerve of one side with an hemiplegia of the opposite side, be called the "syndrome of Weber?" Who is Weber? and why is he advertised by the pathology of the mid-brain? Are we concerned with Weber or with pathology? What scientific conception attaches to the term? Charcot, we recall, used this objectionable term in a clinical lecture a few years before his death, but this is no reason why his followers should use it. This system of nomenclature should be abandoned, and in cases in which it is not abandoned it should be condemned.

Brissaud has also an interesting chapter on the reciprocal relations of hypertrophic cervical pachy-meningitis and syringomyelia. This subject has already been treated in America, and these relations pointed out in patients in the nervous clinic of the Philadelphia Hospital. The publication,<sup>1</sup> embodying these observations, antedates this work of Brissaud by almost one year.

Among other subjects treated by the author, to which we can merely allude, are paralysis agitans, exophthalmic goitre, and myxœdema and cretinism.

The book is remarkably devoid of studies in hysteria. This is notable in a work issuing from the Salpêtrière, and may possibly enhance its value in the eyes of American and English neurologists, among whom, unfortunately, the systematic study of the great neurosis does not appear to be popular. Charcot, towards the end of his life devoted

<sup>1</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, JUNE, 1894.

much time to hysteria and its cognate hypnotism. We do not think, however, that the last word has yet been spoken on these important themes, and we hope sincerely that Brissaud will not let them drop entirely. An English writer has said recently that "hysteria is a by-word," and so it is in England for all that English observers have contributed to rescue it from such an ignominious reproach. Nevertheless it is a living reality, for which the French have done more than all others by giving it the place in nosology which it inevitably must forever retain.

We welcome Brissaud's work as a valuable addition to current neurological literature, and as a worthy product of the clinic from which it issues.

J. H. L.

ON THE RELATION OF DISEASES OF THE SPINAL CORD TO THE DISTRIBUTION AND LESIONS OF THE SPINAL BLOOD VESSELS. By R. T. WILLIAMSON, M.D., M.R.C.P., etc. H. K. Lewis, London, 1895.

There is every reason to believe that the distribution of the blood vessels in the spinal cord will soon hold as important a relation to the diseases of this part of the central nervous system, as the cerebral blood-vessels have long since held to diseases of the brain. In view of the importance of the subject Dr. Williamson has done well to reprint for wider circulation several papers which appeared in the *Medical Chronicle*. The author has stated very clearly the peculiarities of spinal blood supply as developed by the researches of Kadyi, Obersteiner and Adamkiewicz, and has made liberal use of the studies of Marie, Siemerling, Goldscheider, Gowers and others in support of views showing the dependence of poliomyelitis, of multiple sclerosis of transverse myelitis, and spinal syphilis upon the vascular conditions of the spinal cord. To these studies the author adds the reports of several spinal cords examined by himself.

The following are some of the points brought out in these lectures :

The transverse section of the cord may be divided into three arterial districts; 1, the inner part of the gray matter (with the exception of the posterior horn and the caput) which is supplied exclusively by the anterior median artery; 2, the superficial part of the white matter which is supplied by the peripheral arteries; 3, the peripheral part of the gray matter and the adjacent white matter which receive arteries from both the peripheral and central systems. Bifurcation of the anterior median artery at the bottom of the anterior median fissure is rare (Kadyi), which may help to explain the frequent unilateral character of the symptoms in poliomyelitis.

"Whilst the vascular distribution in the spinal cords is unfavorable to the occurrence of hemorrhage and embolism, it appears to be distinctly favorable to thrombosis." This view is supported by the report of an interesting case of spinal syphilis in which the blood vessels of the cord proper are thrombosed. The cord has a further interest since it proves that syphilitic diseases need not begin in the meninges. The writer adduces further evidence in support of the vascular origin of multiple sclerosis, a view that has obtained general credence in connection with Marie's theory of the infectious origin of multiple sclerosis.

The author is also in line with recent doctrines in considering the vascular origin of locomotor ataxia. "The changes in the cord in tabes are secondary to disease of the posterior nerve roots. . . . According to Obersteiner and Redlich, the disease of the posterior roots commences just at the point where they pass through the pia mater to enter

the cord and is the result of a chronic meningitis and arterial sclerosis at this region. . . . In the locomotor ataxia the changes in the cord, and in the posterior nerve roots also, occur first and are most marked in the lumbar region in the majority of cases. . . . This is the region most feebly supplied with blood. Further, the blood supply to the posterior part of the cord and posterior roots is probably feebler than that of the anterior parts."

The lectures are written in a commendable, direct, fashion. The argument is sound. To be sure, there is danger of over-stating the case as with regard to poliomyelitis, for instance. Distribution of the vessels alone will not help to explain all the symptoms, the widely distributed paralysis in the initial stages and the rapid recession and final limitation of the paralysis in many cases. The illustrations are serviceable.

B. SACHS.

THE EYE IN ITS RELATION TO HEALTH. By Chalmer Prentice, M.D., Chicago. A. C. McClurg & Company, 1895. 210 pages.

The title of this book is misleading, as the contents treats particularly of the eye in its relation to disease. It is evidently intended to attract the attention of the gullible public. No educated experienced physician would be willing to accept the assertions and doctrines therein contained, which are manifestly based upon an erroneous diagnosis, and an apparent ignorance of pathology.

The first sixty-eight pages are taken up by an elementary dissertation on the importance of "eye-strain" in the causation of diseased conditions, and in endeavoring to demonstrate that the normal action of every organ is directly dependent upon its motor nervous innervation.

The greater part of the remaining portion of the book contains a detailed report of twenty cases "cured" by either "repression" of vision by means of strong convex lenses or prisms, or by tenotomy of eye-muscles, or by all of these measures combined.

It is, however, magnanimously admitted, that "some cases are beyond or outside of its influence," and that "the failures are due to several causes, especially neglect to follow treatment."

In this report there are alleged cases of diabetes mellitus, melancholia, consumption, paralysis agitans of forty years' standing, and progressive atrophy of both optic nerves. Then follows a tabulated series of fifty-three cases, four being "very much improved" and the others "cured."

Among the latter are the following: Insanity of five years' duration (two cases); Bright's disease of two years' duration; paralysis, hemiplegia of twelve years' duration; cirrhosis of the liver of twelve years' duration; paralysis agitans of ten years' duration (two cases). This is certainly beyond the most extravagant claims of the ultra devotees of "tenotomy" as a panacea. *Mirabile dictu*, the author does not claim to have resuscitated the dead. He seeks to circumvent and anticipate adverse criticism, in stating that "some months, or even a year and more ought to be spent in the experiments suggested in this work," and further adds, "for appreciative and valuable criticism, I turn to those only whose conclusions are always based on a thorough investigation of facts." Who is going to investigate his "facts" as to diagnosis of the disease previous to treatment?

No unprejudiced competent observer would hesitate in relegating these cases to the large class of "faulty methods in observation."

W. M. L.

## Correspondence.

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*To the Editor of THE JOURNAL OF NERVOUS AND MENTAL DISEASE :*

Sir.—In the July number of your JOURNAL, under the head "Duplex Personality," etc., by Dr. R. Osgood Mason, page 422, I read the following astonishing statement anent "subliminal" possession of "supra-normal perceptive powers."

"Alleged facts of this kind are not received by the strictly physiological school of psychologists."

I write to inquire whether this is a "fact"?

Have we among us a "school" of psychologists who are not willing to receive "facts"?

No one can be more interested than I am in this subject of multiple personality, but I should require "facts" of an uncommonly well attested kind before I could believe the possibility of "visual perception gained where vision by the physical organ of sight is impossible," although Dr. Mason assures us that this "is just as well established as any fact in nature" (*sic*).

Certainly, if I rightly understood the attitude of my contemporaries, there is no disposition to taboo "facts" of any kind, even though theories previously formed should be upset. What is most ardently observed is light, especially on these vexed psychological questions.

By all means let those who may be especially hit by the allegation of Dr. Mason hasten to purge themselves of the imputation, for an unwillingness to receive "facts" argues an unscientific mental attitude.

E. P. HURD, M.D.

Newburyport, Mass

THE  
**Journal**  
OF  
**Nervous and Mental Disease.**

AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-first Annual Meeting, held in Boston, June 5, 6  
and 7, 1895.*

*(Continued.)*

Dr. CHARLES K. MILLS opened a discussion on the subject of

CORTICAL LOCALIZATION IN THE LIGHT OF  
RECENT RESEARCHES INTO THE MINUTE  
ANATOMY OF THE CORTEX.

He said that the different theories as to the separate cortical localization of movements and of cutaneous and muscular sensation, which had been the subjects of so much controversy, have again become prominent in the light of the researches founded upon the methods of Golgi, and particularly of those made by Ramon y Cajal, Von Gehuchten, Schäfer, Andriezen, and others. Those who contend against the doctrine that the Rolandic cortex is a purely motor region, believe that they have received additional support for their views. The varying hypotheses with reference to the functions of the cortex were reviewed. Dr. Mills held that, as shown by Forel and Nansen, we have been too long handicapped by prevailing ideas of cell-action and by theories of the parts played by the cell-bodies as originating centres. Impulses are transmitted and transferred by processes as well as by cell-bodies, and the function of the latter is chiefly trophic. The new researches and theories, he

believed, did not compel abandonment of former views as to special localizations, although different standpoints might in some instances need to be taken. Disregarding theory entirely, he believed that the subdivision of the cerebrum into physiologic lobes—higher psychic, motor, sensory (meaning for the representation of cutaneous and muscular sensations), visual, auditory, olfactory, gustatory, and naming—remained for the practical purposes of the physician and surgeon the best. While the whole of the cortex in some of its strata may be regarded as a sensory expanse, the Rolandic portions and particularly the convolutions cephalad of the central fissure, constitute a region that is related to specialized movements of various parts of the body. One calls it motor, another kinesthetic, another sensorimotor, and another executive; but for the purposes of the physician and surgeon it is a motor sphere, the irritation of which causes specialized movements, while its destruction impairs or abolishes these movements. He did not believe with Andriezen that it is necessary to regard the ambiguous and great pyramidal cells of this region, whose apical processes received the terminals of the fillet-radiations, as the first sensory cells of the cortex. Indeed, he regarded it as important to rid ourselves entirely of the idea of sensory cells and motor cells. The cortex contains localized areas. To abandon separate sensory and motor localization would, he believed, necessitate the abandonment of visual, auditory, gustatory, and other subdivisions of the cortex. The cerebral sensory area—that is, the area of representation for skin-sensations and muscle-sensations,—from his point of view, would be that part of the cerebrum where the fillet-radiations in their most compact forms are nearest to the surface of the brain, and therefore this region might continue to be described, as it had been by him, as in the postero-parietal, quadrate, and fornicate convolutions. Destruction of this region, especially if bilateral, caused more or less impairment of sensation. He referred to cases as confirming this view. In the cerebrum, as in the spinal cord, were fields of conjunction between so-called cortical areas, and lesions of these fields of conjunction or at the terminations of the sensory projection-fibres might give rise to temporary sensory disturbances, but these were persistent only when the lesions involved the convolutions included by him in the general sensory area.



## DISCUSSION.

Dr. DANA.—Dr. Mills and I have discussed these questions several times before, and I don't suppose we shall be likely to change our views very much. The doctor has gone over a list of authorities who hold different views as to the relations of the sensory and motor functions of the cortex, and has referred to some advances in anatomy; but, it seems to me, he has not referred to or tried to explain the facts which establish the proposition that I hold regarding this subject. Two or three months ago I went over it again. I collected a large number of other cases and added some of my own, all supporting my view; and it is because I cannot explain the clinical facts or the pathological facts by any other hypothesis than by supposing that the motor and sensory functions are practically united that I still hold to that view. Dr. Mills gives a very learned discussion about the nerve cells and some theories about their relations, but if he will explain to me how it is when I cut away a section of the mid-central or pre-central convolution and find tactile anæsthesia and muscular anæsthesia the next day with paralysis, if he will explain that on any theory except that these two functions are united in that spot, I should like to have him do it. Those cases have become so numerous that it is not fair to get up and talk about this subject without referring to it. Why doesn't he explain those cases, and then we will try and accept his theory if he can adjust that fact. Such cases I have had, and so have Drs. Knapp and Putnam. They are extremely numerous in the literature of neurology now. If you carefully shave off the anterior, pre-central convolution in this middle part, you will get sensory disturbances, anæsthesias.

Again, Dr. Mills perhaps may not be aware that in the living subject, two persons, an English physician, Dr. Russell, and myself have directly irritated the motor cortex, also in front of the fissure of Rolando and produced sensory disturbance associated with motor. Now, if a man's sensory functions are located in the gyrus fornicatus, I do not understand how the passage of the electric current applied to the arm-cells should cause sensations entirely analogous in the arm to those of a tactile impression made upon the skin. In fact, the whole weight of clinical evidence is in favor of motor sensory union, the surgical operations, the tumors, the

softenings, the laboratory experiments all furnish facts which cannot be explained on any other hypothesis, so far as I know, than that the two functions are essentially identical anatomically.

Dr. Mills has a sort of argument from analogy. He says the other special centres, the centre for visual sensations and for auditory and gustatory sensations are separate from the motor centres, and that therefore cutaneous and muscular sensations would naturally have centres separate from the motor areas. I admit that that is a plausible theory, but it does not at all hold against any actual facts, and when we come to analyze it, it is not plausible after all. The visual sensations are not so closely related to motions as are the cutaneous and muscular sensations. We have got to have for the performance of co-ordinated movement a very close anatomical relationship between the muscular and cutaneous sensations. We cannot afford to have muscular sensations impinge on the gyrus fornicatus and have associating fibres then conduct these semi-impulses to the central convolutions. They must be close together anatomically for the purposes of a perfect machine, otherwise we could not make rapid motions; and I believe it is for that reason that nature has gradually shifted the sensory receptive cells, so that they are in approximately close relation with the motor cells.

I believe that perhaps if we were all agreed as to exactly what is meant by sensations and exactly what tests we use for making sensations, and were agreed about the terminology of our discussions, we would not have perhaps so much discussion about this matter. It may be we are not so far apart after all. I quite agree with Dr. Mills that sensations are not closely localized, and I agree that we should speak of sensory areas, but I do not believe we have sensory areas separate from the motor ones back in the post-central and parietal lobes or in the gyrus fornicatus. I do not accept Dr. Starr's view, that the sensory centres are back of the fissure of Rolando, for from the evidence I have been able to collect, there is just as much proof that the pre-central convolution has sensory functions as that the post central one has.

Dr. PUTNAM.—One or two things only I should like to say. In the first place, I think Dr. Dana's closing sentence must be accepted as absolutely certain, that the convolutions in advance of the fissure of Rolando, the

function of which we ordinarily associate with localized movements, have also to do with sensation. In support of that fact I may mention one case, among many, where Dr. Warren excised a minute piece of the cortex in a young man with epilepsy beginning in one hand,—a piece a few millimetres in diameter,—with the result of producing paresis of the hand, paralysis in the beginning, and also a disturbance of the sensibility of the whole hand, which, however, soon passed away. It seems to me that shows that in some way the function of sensibility is closely related to the same centres that represents the function of motion. What that relationship, is it seems to me, is a matter still calling for a good deal of study. My own view of the difficulty is that we are still decidedly lacking in a sufficiently adequate physiological or psychological conception of what we mean by those various terms. My own view is that the function of sensibility must be very widely distributed. When any part of the body is touched or any motion takes place there, various other functions are immediately incited to action. A motion of the eyes must follow very rapidly on a sensation, because the individual touched must immediately know from what cause the touch arises, and the like : so that the close relationships must be established, and as a matter of fact, we have every reason to think they are established, with all sorts of cerebral functions of various kinds. Another thing which is interesting, I think, is that when we apply a sensory stimulus we are applying what may perhaps be a very gross molecular disturbance of a nerve at the part touched. This then impinges on some part of the brain primarily, and from there it must spread widely, and passing through some secondary channel if its natural channel is obstructed. It is instructive to remember that a sensation will make its way from a minute portion of the substance of the spinal cord that is left, so in the case of the brain, if one channel is cut off it will make its way into a dozen other channels. A case illustrating that occurred at the Massachusetts Hospital. Dr. Richardson has recently operated on a patient I saw first in consultation outside with the result of eventually removing by a second operation an enormous tumor larger than an orange, which lay directly in the motor tract. The patient had been paralyzed for some time, and it would certainly be that the whole of the motor area had been destroyed by the growth with a good part

of the internal capsule. Nevertheless that patient still has a very considerable degree of sensation left, though, of course, modified. Contact gives rise to a painful distressing feeling, but pricking and even a slight rubbing is felt and the localization is still fairly good, although contrary to what Mr. Horsley says, he localizes not nearer the root of the limb where the contact is made, but farther from it, towards the extremity of the limb; nevertheless he feels in a very high degree, so that although the removal of the motor convulsion has affected the sensibility of the limb it has not by any manner of means destroyed it, and that it seems to me is a thing we have got to bear in mind. We know nerves will stand a high degree of injury sufficient to entirely destroy their motor functions without interfering materially with their sensory functions. The same thing in the cord; a minute portion of it will convey centripetal impressions. Sensibility would seem to be rather peculiar in the fact that it is almost always related to something else. A sensation is felt almost always for the sake of something else.

I would like to say in addition that I have in mind one or two cases where the sensory functions have been disturbed in connection with paresis of one hand, and where it seems to me that I could detect a higher degree of impairment of sensibility on the side of the ulnar part of the hand than on the side of the median portion.

Dr. STARR.—I think that it is our business, if possible, to bring together all the various facts that come into our knowledge in regard to this question. Now, we certainly have facts that Dr. Dana has stated that are indisputable, and those of us who are constantly seeing cases of cerebral surgery and excising cysts and portions of the cortex, small in extent, or who are seeing pressure produced upon the cortex by clots that can be removed, are perfectly positive that a lesion of a small limited area of the so called motor zone does inevitably produce in almost every case more or less disturbance of sensation. I do not hesitate to say that I consider that I was completely mistaken in 1890, when I maintained that sensations were received only behind the fissure of Rolando. I believe with Dr. Dana to-day that there are disturbances of sensations produced by small lesions anterior to the fissure of Rolando, and that the sensory area of body corresponds exactly with the motor area of the body so far as we can determine clinically. I cannot shut my

eyes to the facts that have been brought out time and again right before me on the operating table, and subsequently to it, and I do not hold to theoretical explanations when in contrast to the facts we can put our fingers on definitely, but I think it would be a mistake to draw too broad a conclusion from these facts. That is one set of facts we have got to regard.

Secondly, when you come to analyze the embryology of the brain, it seems to me that another set of facts confront us. The recent address of Flechsig throws a great deal of light upon this subject. You probably all have read his address translated in the *British Medical Journal*, April, 1895. Flechsig showed conclusively that in the course of development there are two distinct and separate periods of development to be traced in the embryo in the cortex: the first period in which there are formed projection tracts of different kind, the so-called motor tracts; and sensory tracts of distinct kind, visual, auditory and olfactory, and also tactile, the latter are distinct and separate from the motor and they terminate in areas of the brain separate from the motor and posterior to the fissure of Rolando. That is the second set of facts. We cannot get away from these facts of Flechsig. He shows that the second stage of the development of the cortex is an enormous development of association fibres, separate and distinct in the life of the child because it is only when these fibres begin to develop that evidence of the combination of the various sensory memories are shown in conscious intellectual acts. That is the second set of facts, and I think the true solution of these apparently contradictory facts is indicated by Dr. Putnam in his phrase, which I like very much, the diffusion of sensations, and I think that there we have the explanation of the whole problem. We know from Cajal and Golgi methods of staining that the sensory fibres, when they come in, do not terminate in a distinct sensory cell. And I think we must give up our ideas of sensory cells as such. When you think of the innumerable sensations that come from the distinct touch as of a cigar on the finger, the reflexes it occasions, the reflex withdrawal of the finger, the vasomotor reflexes and the automatic acts, such as catching one's breath, and then, lastly, the reception of these conscious sensations in the cortex, I think it perfectly evident that that term diffusion of sensibility is a capital one, and I think our present anatomy shows why it is; it is because these sensory fibres terminate in

brush-like expansions, so that we must conceive of a sensory fibre as virtually terminating in a long region of the nervous system, in fact, almost throughout its entire length. Now suppose that with the continued passage of sensations over these fibres diffusing themselves in general directions habit opens the way in one direction rather than in another, that a sensation coming in might be diffused from the arm over the entire parietal arm centre and also diffused over the motor centre, but inasmuch as we guide our movements by touch habitually, the result that a greater passage is opened out over to the motor zone, and therefore sensations reach the motor zone before and more rapidly than they reach the real sensory zone, and they are received there just as decidedly as they are elsewhere. I think that diffusion of the sensibility in various areas gives an explanation for the fact that a few cases are on record where we get sensations lost alone, and the vast majority of cases show that sensation and motion are lost together. The sensations go in different directions, but habit produces the effect of sending them chiefly to the motor area, and therefore they are received there as well as elsewhere. From Flechsig's researches I think we must abandon the idea of consciousness and conscious perception, and perhaps to some extent of conscious motion being associated with particular cells. It seems to me it is just as reasonable to believe it is associated with association fibre action, the combined activity of various areas of the brain, as it is with single cells, and I think we will come down finally to the fact that a cell is to be regarded more as a trophic centre than as a motor or sensory cell.

Dr. DERGUM.—I am glad to see the stand that has been taken—so different from what was taken a few years ago with regard to cerebral localization. It enables us to explain certain other phenomena not alluded to here, namely, such facts as that the fact that the cuneus fails to atrophy in cases of blindness lasting for many years. Certain it is that we have no right to conclude that the cuneus is related to no other function than that of vision. It is probable it does many other things. To my mind the various centres of the cortex, as we know them clinically and pathologically, are simply highways of ingress and egress to the general cortex. The facts give us no right to say that this portion of the brain does only this special thing and that portion only that special thing. A so-called sensory centre is merely a

point at which a stimulus reaches the cortex, and from which it is diffused through neighboring and perhaps distant areas. General biological considerations also would negative the sharp differentiation of cells into special functions. Nerve protoplasm reacts to certain forces ; to say that one cell reacts to one mode of motion, and another to another, is to my mind unphilosophical and not borne out by general biological considerations.

Dr. RICHARDSON.—I have nothing special to say except that I have been extremely interested in this discussion, although I know very little about the subject except from the clinical standpoint. I would like to say with reference to the tumor which Dr. Putnam spoke of, that so far as I know, there was no destruction of the motor areas except by pressure of the growth. The tumor was very large, more than filled a pint-jar ; the pathologist thought it would weigh more than a pound, so that it took up a very large portion of the left hemisphere. During the operation I touched the falx throughout a great deal of its extent, my finger passing through the very large opening through the *cristi galli* to the tentorium. But so far as I know, there was no destruction of brain tissue by manipulation during the operation. The man is now doing very well.

Dr. JOSEPH COLLINS.—I have recently had a case of cerebral surgery in which development of sensory and motor defects have been rather peculiar, and I would like to ask if any one of the members had seen such an occurrence. A young man had his first attack of Jacksonian epilepsy in November, and the movements were contraction of the finger and thumb. When I saw him in March he had had three attacks, only one of which had been associated with loss of consciousness. At the advice of Dr. Sachs and myself, Dr. Gerster operated on the patient, making the opening over the superficial cortical thumb and index-finger centre. It was with a great deal of difficulty that we got through the skull. The patient bore the anæsthetic very badly, and we had to postpone the operation after the skull had been opened, that is, we had to postpone opening the membranes and excising some of the cortex. That night the patient had an extremely severe attack which had been preceded a short time by the development of very high temperature. The attack was accompanied by loss of consciousness for a long time. The following morning he had paresis of the right upper extremity and loss of sensa-

tion, that is, loss of tactile sense, loss of sense position and loss of muscular sense in the right hand. Three or four days afterwards we opened the dressings and found a good deal distention of, and effusion into the surrounding cellular tissue, but otherwise where the operation had been done, it seemed very much like normal. Then Dr. Gerster opened the dura, and cut out a large part of the cortex, and incidentally I might say, we found an old pachymeningitis there, and the Nissl stain revealed degeneration of the cortical cell. We put him back to bed, and tested his hand for sensibility that evening, and found that the disturbances of sensation had all disappeared, every one of them. Now, it seems to me that that opens up an avenue that has not been recognized, inasmuch as the disturbances of sensation came on before the cortex had been excised and disappeared almost immediately after excision of the cortex. If we put the localization of sensation and motion one over the other, superimposed in the paracentral convolutions, this experience seems to indicate that they are not intimately blended, because when we excise the motor cells the sensory anæsthesia disappears at once, while the paresis remains.

Dr. ANGEL, of Rochester.—I can corroborate Dr. Collins because I had a similar case. I made the prediction that there would be paralysis for five or six days at least, and to my surprise when the boy recovered from ether two or three hours after operation, I tested him thoroughly and found sensory paralysis. Within twenty-four hours there was absolutely no sensory paralysis or sensory disturbance. It seems to me that that is the characteristic distinction between sensory disturbance from removal of the cortex anterior to the fissure of Rolando and posterior to the fissure of Rolando. I think you will get a deeper and more prolonged disturbance of sensation posteriorly than anteriorly. It seems to me that must be explained possibly through the association tracts. I do not believe it is possible to make an impression upon any centre of the brain closely related to another centre without affecting temporarily that sensory centre. Whether this will explain the reason why we have a short paralysis of sensation when we remove the motor cortex or not is, of course, beyond our power yet to determine. It may be by cutting into the cortex that we affected the rootlets of the radicals from the sensory tract which we suppose reach over and produce this diffusion of sensation that has been referred to.



Dr. PRINCE.—I would like to say one or two words only on this subject. It is one in which I have followed the evidence for a long while with care. I agree entirely with Dr. Starr, that we must take into consideration the facts, but they must be all the facts and not an expurgated edition. Any theory to be acceptable must explain all known facts and not skip those that inconveniently present themselves for examination. Now Dr. Starr has insisted upon two sets of facts, namely, one set, that following operation on the motor cortex you frequently get some loss or disturbance of sensation; but I wish to call Dr. Starr's attention to the logical fact, that it is an inference from the clinical fact and not the fact itself that these portions of the cortex are the centres of sensation. This inference is of value only so far as it explains all the other facts. The other fact to which he has called attention—the distribution of the fibres is an interesting one and of value as evidence. But there is a third fact which Dr. Starr has left out of consideration, and that is the fact that in many cases which have been referred to by other gentlemen present, there has been no loss of sensation. These cases have been numerous. If I remember correctly the figures of a large number collected by Charcot in which the presence or absence of anæsthesia was noted, in a majority no loss of sensation was present. Some will say that these cases were imperfectly observed. Well, then, suppose that only a minority presented no sensory loss. Those who have read Darwin's autobiography—a book which everyone enjoyed in scientific research should read—will remember that Darwin in a certain passage attributes his success largely to having always recorded the exceptional cases, those that did not follow the general rule. He thus was enabled to formulate a general law to which all cases would be subject. If we are to draw correct inferences from the facts as to the localization of sensation, the minority of cases must be explained. Several cases have just been mentioned in this discussion, where excision of a portion of the Rolandic cortex has not been followed by sensory loss. A great many could be mentioned. As I have said, I believe that the majority of such cases do not exhibit sensory loss, but I assume it to be the other way. I wish to refer now to two cases which are of great value as evidence. Many gentlemen will remember the case of Dr. Hale White, published in 1893, in which case a portion of the motor cortex was excised, and the sen-

sation over the corresponding part of the body afterwards tested with great care with the only purpose of determining the relation of sensation to the motor cortex. Although paralysis followed there was not loss of sensation.

But what seems to me a clinical test is a case published lately by Beevor and Ballam, *British Medical Journal*, January 5, 1895. In this case it will be remembered that a piece of brain in the Rolandic area was scooped out, equal in size to "half an orange," as described in the report. A hole two inches in depth was made involving a large portion of the ascending convolutions, the quadrate lobule, and a portion of the adjoining parietal convolution. Before the operation there was hemianæsthesia with paralysis. After recovering from the operation there was persistence of paralysis, but there was absolutely no loss of sensation whatsoever, although the most minute and careful tests were made. A case like this seems to me a clinical one. If so large a portion of the motor cortex can be destroyed without resulting loss of sensation, I am unable to understand how this portion of the cortex can be spoken of as a sensory centre. I admit there is a good deal of ambiguity about the meaning of the word "centre." But for practical purposes and within the meaning of the discussion we must mean a place in the cortex, the destruction of which abolishes certain functions. Now these cases, cases like the ones I have cited—and they are numerous—must be taken into consideration. They cannot be bound over as disagreeable cases which we don't want to hear. They may be unpleasant but they are true. Dr. Dana asks how, then, are we to explain the other group of cases, in which loss of sensation does follow lesions of the Rolandic cortex. That is another question. We may answer that we don't know how to explain them. But because you can't find a satisfactory explanation you have no right to draw inference which are illogical, and leave out of consideration a large group of contradicting facts.

If asked to present a theory, I think I could offer several that would satisfy the conditions, although I have no proof of the correctness of anyone. What is needed is evidence. Here is an explanation which is not impossible.

We know very well that we may have an injury in other parts of the brain, followed by disturbance of function which we know is not localized in that part; for

example, it is common to have injury of the corpus striatum with aphasia and sensory disturbances; even temporary hemianopsia is common with lesions of the corpus striatum, and yet I don't suppose any one localizes the visual centre in the corpus striatum. Aphasia is common as a secondary disturbance with lesions in different parts of the brain, and yet we know that motor aphasia depends upon a specially localized lesion. We, therefore, must regard such phenomena as secondary disturbances. In the case of the corpus striatum we can explain the symptoms by presence of œdema or local congestion of the internal capsule. It is possible that such secondary symptoms may sometimes be explained by some form of irritation of associated fibres or the effect of inhibition.

The observation of Head have placed the symptomatic effect of reflex irritation upon a new basis, and offer a new field for research. But whatever the explanation, we must not hastily accept inadequate theories simply to cover up a hiatus in our knowledge.

Dr. PUTNAM.—I wish to reinforce what both of the last speakers have said. We have in the relation of motor and sensory aphasia a very pregnant example of what has been referred to. No one would speak clinically or anatomically of the sense of hearing and comprehension of spoken words as located in the same part of the brain, but we know these two functions lean to one another internally so closely that not only we get more or less aphasia from disturbance of the centre for hearing, but we also get (to a less degree) disturbance of comprehension in most forms of motor aphasia; in short, cerebral functions do not exist for themselves, they exist as related to other functions, and you cannot destroy one without more or less impairing others, although it may be only for a brief time.

The CHAIRMAN.—When Dr. Mills wrote to me the question of recanting his heresies, he asked me to take part in the discussion. I have very little to add to what has been said. It seems to me that the whole of our knowledge of the neuron goes to show the very pronounced dependence of the motor neuron upon the sensory neuron. In the primary neurons it has been clearly proven that the terminal processes of the axis cylinder of the sensory neuron are closely connected with the apical processes of the motor neuron in the cord. Golgi in his earlier work held clearly that we cannot make a

distinction in the cortex from its minute anatomy as to what portion of it was sensory and what portion of it was motor; in other words, that the cells which he said at that time were motor were to be found in the areas of the cortex distinctly called sensory, that the cells considered as sensory were to be found in the area of the cortex which he considered motor. That, of course, is not to be accepted without more or less question; but we do know this, that the recent studies in the development of the fibres in the brain itself, show that the sensory tract passes distinctly upwards toward the central convolutions, the parietal region, rather than downwards and inwards toward the gyrus fornicatus, where Ferrier sometime ago located the sensory centres, and where Dr. Mills, I think, still has a leaning toward placing it. In other words, judging from all the analogies in the anatomy of the brain, the higher sensory neurons must either pass upward directly to connect by their axis cylinder processes with the cell bodies of the motor neurons, or there must be association tracts from these axis cylinders going up to the motor neurons. Now we find no association tract passing up from the gyrus fornicatus to the central fissures, and furthermore, although Dr. Prince has just brought up the negative cases of lesion of the central convolution with injury to sensation, there have been in the past, as Dr. Prince will certainly remember, a very large number of so-called negative cases about the central convolution with absolutely no motor disturbances. Most of those with our increasing knowledge have been explained away, and ten negative cases do not, it seems to me, afford as much evidence as one positive case. The positive cases of sensory disturbances following lesions behind and in front of the fissure of Rolando are increasing in number and become, as it seems to me, very conclusive. The positive cases of lesion in the neighborhood of the gyrus fornicatus are very few and far between. As Dr. Dana has spoken of my own case, I might refer to it again, saying that that was one of the cases of superficial, very slight lesion of the central convolution that was followed by a very distinct loss of sensibility. I would like to speak of the case briefly because I encountered it the other day in such a form that if my name had not been connected with it, I should not have known my own case. Brissaud in his recent work alluded to the case as one where the finger penetrated deeply into the cerebrum almost to the capsule.

DR. CHARLES K. MILLS.—I have at least succeeded in doing one thing, perhaps two; in the first place, in bringing out an interesting discussion; and, in the second place, I believe, a discussion, which, on the whole, confirms the standpoints I have always taken, strange as it may seem to the gentlemen present. It seems to me astounding that Dr. Dana and others speak of physiological or pathological lesion of the motor cortex as exceptionally producing motor symptoms alone. Every one of us must admit the cases referred to by Dr. Starr, Dr. Dana, Dr. Putnam, and Dr. Knapp, because we know they have been well studied and well recorded—cases in which sensory phenomena of some sort have accompanied the motor phenomena in cases of destructive lesion of the pre-Rolandic cortex. We must admit, too, the two or three cases—a miserly number—in which certain experiments upon the cortex in life have caused peculiar temporary disturbances of sensation at the time. It was my purpose not to refer in detail to any cases, but simply to open this discussion. Against these cases of sensory disturbance for strictly limited lesion of the motor cortex, the cases in which symptoms were motor alone instead of sensory are as one hundred to one, and we do not think it longer worth while to collect these cases. I need only refer to my own experience, because it would take too long to bring up statistics. In a score of these cases with the greatest care patients have been examined in life, and the lesions located in death, and in cases of operation the greatest care has been taken in studying sensory phenomena, and those sensory phenomena have not been present. I might mention two cases of my own, one in which Dr. Hearn clearly excised a portion of the cortex right across and in front of the Rolandic fissure; another case in which Dr. Keen did for me the same thing, and in which we studied with the greatest care, as soon as it was possible; the sensory phenomena were not present, but motor phenomena persisted day after day, and day after day changes were noted until the patient was largely restored as regards motion. Others saw the case, and although a large area of the Rolandic cortex had been excised no sensory disturbance resulted. It is not worth while to pit these cases which Dr. Dana has with great skill and labor collected, as an argument of great value against the very numerous cases which teach the other thing. Certainly, however, such cases must be acknowl-

edged. I believe the cases are as stated. Have we no explanation for them? These cases arrange themselves into two or three classes. In the first place a large number of them are temporary; all will admit that, I think. The disturbances, in the second place, are sometimes purely subjective, first admitting the temporary objective disturbances of sensation. Thirdly, they are, as in some of the cases reported by Dr. Dana with great care, sometimes persistent. Charcot before his death suggested one explanation, namely, that these cases are somewhat of the nature of the cases in which we have sensory disturbances in hysterotraumatism. Nothing is wrong about that explanation. Many times we have been told about hemianæsthesia, and about segmental anæsthesia, as the result of a man being struck on the head or leg or trunk, and certainly the traumatism inflicted by the surgeon's knife, or by injury or disease in acute cases is greater than that which we have in other cases. Another explanation is that which I suggested in 1888, which also grows out of these later researches in cortex anatomy. Undoubtedly every area of the cortex, visual, auditory, gustatory—sensory in my sense—must be related and anatomically connected with the motor regions of the cortex, for we have auditory-motor and visual motor phenomena. It may be that through the destruction of the terminals of the fillet radiations, where they touch the apical processes of the pyramidal cells, or of the cortical field of conjunction between the sensory and motor areas, that temporary disturbances of sensation in some instances result.

I protest against the assertions about positive cases on one side and negative on the other. It is just the other way so far as the record of cases and experiments are concerned. The remarks made by Dr. Starr are, I think, on the whole, confirmatory of what I have stated here. It would seem to me, therefore, Mr. President, that the weight of argument, and the weight of evidence, is still in favor of the localizations to which I have always clung, and which were believed in by Charcot, Ferrier, Bechterew and others. I wish to be understood clearly. Dr. Knapp in closing spoke of the gyrus fornicatus. I have never claimed that the gyrus fornicatus was the sole region of common sensibility in the cortex of the brain. My own position always has been that the sensory cortex proper includes at least a portion of the gyrus fornicatus, quadrate lobule and posterior parietal

convolutions, and the arguments I advance must stand for this region and not for part of it.

Every neurologist knows we have as a hundred to one, for physiological reasons, lesions occurring in the motor region of the brain as against lesions occurring in the sensory region. It is very rare to have a lesion of any sort—tumor, hæmorrhage, or softening or any of the common lesions of the cerebrum—in the gyrus fornicatus, quadrate lobule or the posterior parietal region, as compared with the immense number of lesions in the so-called motor region of the brain, and that is one explanation of the fact that we have so few positive cases to corroborate our views. Another is that probably bilateral destruction is necessary to bring about complete anæsthesia in these cases.

Dr. DANA.—I should like to ask if Dr. Mills will tell us specifically whether he denies that the central convolutions have any sensory function, if he absolutely excludes sensory function from the central convolution.

Dr. MILLS.—I believe what I have said. For practical purposes I believe the central convolution, or at least the convolutions cephalad of the central fissure, have no sensory functions. The posterior central convolution, perhaps, may, with the posterior parietal and the region I have named, take some part in sensory phenomena.

Dr. DANA.—Then I don't think we differ very much.

*Afternoon Session, June 6.*

## REPORT OF A CASE OF TUMOR OF THE CEREBELLUM, WITH AUTOPSY.

BY DR. E. D. FISHER, OF NEW YORK.

Professor of Mental and Nervous Diseases Medical Department of the University of New York.

OPERATION BY DR. JOHN F. ERDMANN,

Professor of Practical Anatomy in Bellevue Hospital Medical College; Visiting Surgeon to the City, Work and Almshouse Hospitals.

**S.** F.—German, 30 years of age, married, domestic. Entered hospital, September 1, 1894. The family history of the patient is entirely negative. Denies specific disease, and is not alcoholic. Was in good health till 1890, when she had an attack of influenza followed by headache. There was also vomiting accompanied by retching. The vomiting recurred at intervals for about one year, not to return again for three years. Headaches have been more or less constant since the original attack of influenza, but at first were noticed only in the morning, later, in the evening as well; till, finally, they became constant. The pain is referred to the brow, extending back to the occiput, and radiating to base of the brain with point of greatest intensity over right orbit. Two years ago, eye-balls became prominent, and flushing of the cheeks appeared. In the spring of '93, was much disturbed by flashes of light before both eyes, more marked on the right. In August, 1893, right eye could distinguish between light and darkness only, although sight in left eye continued good. In March, 1894, she entered German Hospital where she was treated with large doses of iodide without beneficial results.

In May, 1894, patient became entirely blind and headaches became more severe. At this same time, she lost the sense of smell, and became deaf in the right ear



which was subject to "stinging sensations" as well. Speech and deglutition were not affected. Memory slightly impaired, but intelligence undisturbed. For a year before entering Hospital for Incurables, there had been some general weakness of arms and legs.

In the spring of 1894, she had her first general convulsion. These convulsions have continued since then at irregular intervals. Apart from the evidences of nervous diseases already noted, general health had been good, appetite excellent; menstruation regular, and nothing abnormal as regards bladder and rectum.

Her condition on entering hospital September 1, 1894, was as follows:

A woman of large frame, not anæmic, well-nourished, presenting no abnormalities of skin or muscular system. Her general expression is that of one totally blind. Her vegetative organs present no symptom of disease. Pulse regular and full, becoming very much more rapid, however, on suddenly rising from chair. Her intelligence is excellent. She is patient and uncomplaining; hair thick and glossy; eye-balls, prominent; pupils, equal, widely dilated; left iris reacts slightly to light; right, not at all.

Examination of eyes by Dr. Esson showed a double neuro-retinitis with right disk in condition of incomplete atrophy. The arteries are small with thickened walls. A clearly discernible connective tissue formation exists around the arteries. This connective tissue growth extends only one disk's width from the vessel-wall. The veins are somewhat tortuous. The upward movement of the globes is somewhat limited, and they have a slight tendency to turn towards the right. There is a slight divergent squint in both eyes. R. 5 + at the fundus; 2 + at the periphery.

To the upper and outer side of the optic disk is a yellow spot irregular in outline.

L. Patches of atrophic degeneration more marked on nasal side; refraction in centre of disk is 4 +; fundus 1 +. At the periphery of disk, involving about three-fourth of its circumference, the atrophy is more marked than in the disk proper. The amount of atrophy in both eyes is about equal. Patient is completely blind. Complete deafness in right ear, with impairment of bone conduction. Left ear can hear watch tick two inches from meatus; speech is normal; articulation distinct; deglutition and secretion of saliva are normal; taste acute; absolute bi-lateral anosmia; subjective sensations

of a stinging character passing to the frontal sinuses when penetrating odors are given her to smell; there is a very slight paresis of the right facial nerve. The patient is unable to wrinkle her forehead on either side. States, however, that she has never been able to do this, and that her mother also was without that power. There are no paralyses of the extremities; co-ordination is everywhere good; there is absolute absence of intention tremor, twitchings, contractions, contractures, or choreiform movements; gait is slow, hesitating as usual with the blind, but firm. Romberg's symptom is not only wanting, but patient can stand steadily on one foot at a time. She has no subjective sensations of dizziness. All forms of sensation are absolutely unimpaired. There is some slight increase of knee-jerks, with very faint right-sided clonus.

From her entrance into hospital till time of operation the patient's symptoms were slowly progressive. There were several attacks of convulsions general from the outset, accompanied by unconsciousness. The convulsions were short in duration, and the consecutive coma lasted about ten minutes. She was subject to attacks of projectile vomiting independent of time of taking food. Pain became extremely severe, preventing sleep, and commencing at right orbit radiating from the base of brain down through limbs. She was not only willing for an operation, but desired it, and although told that an operation would be but palliative, she said that she would take any possibilities rather than continue in the condition in which she was.

December 9, operation.

There was some pain after the operation. It disappeared, however, in a few days, not to return until a few days before her death. There was never any return of the convulsions until shortly before her death. Three weeks after the operation, hearing on contact returned to the right ear.

About a month after the operation, she could detect odors with the left nostril and the iris light reflex was active on both sides. She also saw, as a shadow, objects held before her eyes, though the truth of this may be doubted as she failed to recognize a lighted candle held before her eyes.

On February 7, patient had a chill and complained of great pain in head and back. Her temperature, which had been normal, took on the irregular course of infec-

tion, the skin around the wound became reddened and œdematus. There were also some convulsions beginning in right face and becoming general, and on the seventeenth she died, having been unconscious for several days.

The interest in the case as reported consists in the absence of localizing symptoms. The general symptoms left no doubt of the presence of tumor of the brain, *i. e.*, the optic neuritis, the convulsions, and the excessive cephalalgia. The situation of the pain was misleading. It had from the very beginning been situated in the right side of the head, with its point of greatest intensity over the orbit. There had, indeed, been some occipital pain, but this was almost lost sight of in the intensity and local tenderness anteriorly. The fact also that the loss of sight on the right side preceded that of the left, and the statement that there was loss of smell on the same side, later extending to both sides, lead me to believe the situation of the tumor as most probably under the frontal lobe and lying over the orbital plate, compressing directly the right optic nerve in front of the chiasm. This was confirmed by the absence of any other cranial nerve lesions which could aid in localizing the neoplasm. There is mention of a slight right facial paralysis, but it was indeed so slight that I was not able to satisfy myself of its existence. The presence, also, of deafness on the right side, which was absolute and which, singularly, after the operation partly disappeared, had suggested a cerebellar tumor. All other motor or sensory symptoms were absent, the hand grasp was equal and excellent on both sides, and there was nothing abnormal in the walk, evidencing the slightest weakness; the muscles were well-developed, and the reflexes approximately normal. At no time was there the slightest tendency to any staggering in the gait, nor any inclination to move or fall to one side, anteriorly or posteriorly. There was no inco-ordination, nor did the patient complain of dizziness or vertigo to any extent.

On these grounds we excluded a cerebellar growth, although we had that lesion in mind.

A second interesting point in the symptoms, was the long duration of the growth without producing any mental deterioration. The patient thoroughly understood her case. She had been examined by a number of physicians and was able to describe her feelings and her previous history with great accuracy, and insisted on an

operation, if there was the slightest chance of being relieved of her intolerable pain.

The third point of interest was the complete relief from pain which followed the operation, and which continued until the symptoms of basilar meningitis manifested themselves.

The autopsy showed the cause of death to have been a purulent meningitis at the base of the brain extending down the cord. This evidently was of recent origin and occurred probably not more than ten days before death.

There was no pus found in the frontal lobe, simply some softening and disorganization from the hernia cerebri. This prevented, as Dr. Erdmann states, a bony union of the bone flap, but as the specimen which I now present shows there was a good fibrous union. The tumor was found to be a glioma occupying the right cerebellar hemisphere, compressing the pons varolii on that side, and including in but not destroying the cranial nerves. The growth of the tumor was very slow for a glioma, and I had been led to expect from this fact that we had to do with a syphiloma.

The tumor was  $3\frac{1}{2}$  inches long and  $2\frac{1}{2}$  inches wide, as shown in the specimen I now present.

This case, therefore, shows of how little value for localization the situation of cephalalgia is when the growth is deeply situated, and slow in its development, and it also demonstrates how tolerant the cerebral structures are when injury to them is gradual.

It is also an evidence of the relief which can be given at a long distance from the lesion from the lessening of cerebral pressure. On opening the skull by Dr. Erdmann, the dura mater was found very tense, and a large amount cerebro-spinal fluid was lost. I at first thought we would find the growth under the frontal lobe pressing it upwards. The considerable manipulation of the frontal and temporal lobes, which was necessary in order to discover the presence or absence of a tumor, did not produce any shock, at least as shown by the course of the case in the following weeks.

The operation was a somewhat protracted one and it was necessary several times to resort to artificial respiration; this I ascribed to the ether, which was badly borne by the patient. I believe, indeed, that the brain can stand considerable manipulation without injury.

The operation was an exploratory one, as no positive

diagnosis was made of the situation of the growth, and as I have said, the localization of the pain was our principal guide, the relief of which was all that we anticipated.

I would take this opportunity also to refer to the method of operation in these cerebral cases by the bone flap. Several such operations have come under my observation with invariably good results. There does not seem in my experience to result any cerebral shock from the use of the chisel and hammer and in cases when suppuration from any cause or hernia cerebri is absent, the bony union is good. In a case operated on for me by Dr. Woolsey and reported by him, the autopsy some two years later showed a perfect bony union, without adhesion of the membranes.

I would take this opportunity to thank Dr. Pearce Bailey for the history of the case, and Mr. Slade of Bellevue College for the illustration.

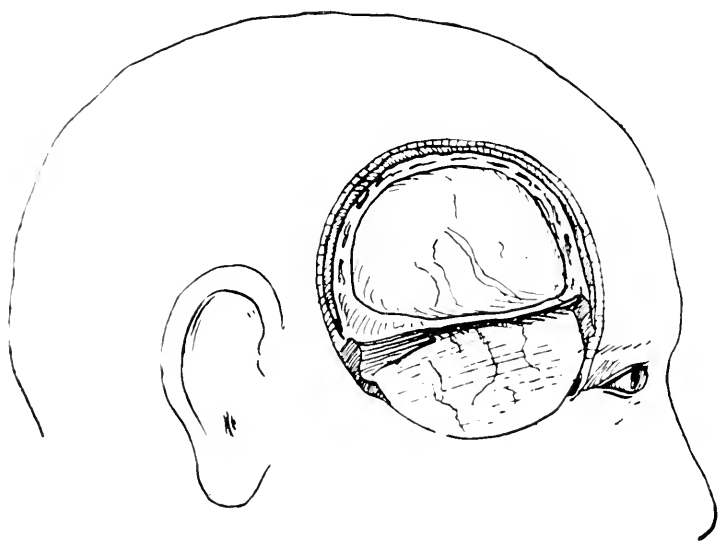
OPERATION DECEMBER 9, 1894, BY DR. JOHN F. ERDMANN,  
IN THE PRESENCE OF DRS. FISHER, BAILEY AND OTHERS.

A large omega-shaped flap was made with the base downwards beginning about three-quarters ( $\frac{3}{4}$ ) of an



inch to the left of the right external angle of the frontal bone and extending backwards for two and a half ( $2\frac{1}{2}$ ) inches. This size opening was made with a view to exposing the anterior and middle fossæ. The flap was of the composite type, skin, muscle, etc., and bone all in one, the bone being chiseled through in the incision made by the knife.

After raising the flap and breaking the bony base line, there was a marked bulging of the cranial contents protected by healthy dura.



Extradural examination of both fossæ was made with a negative result. The dura was then incised, with a view to examining the cerebrum, whereupon fully three-quarters of a teaspoonful of cerebral tissue was extruded. Careful digital examination was then made of the cortex of the anterior and middle lobes without finding any evidence of cortical or deeper-seated new growth. At this stage of the procedure the patient's condition was such that to further search was considered inadvisable. Owing to the marked bulging considerable difficulty was experienced in closing the incision in the dura and

in properly adjusting and retaining the composite flap in its proper position.

A small drain of sterile gauze was introduced, and then after suturing, a firm dressing was applied over a compress of gauze used with a view to holding the bone flap in place. Owing to the succeeding dressings being loosely applied by the attendant in the absence of the operator, bony union never took place, but the flap of bone became united by means of fibrous tissue. A small cerebral hernia was present for about a week, and then union took place in the entire line of the cutaneous portion of the flap, except at the site of the original drain. This small fistula existed at the time of death, eleven weeks later, discharging during life a thin watery fluid.

Autopsy.—Small fistula at lower portion angle of the wound; otherwise complete union in the skin. Fibrous union of the bony flap to the surrounding bone with the bony flap super-imposed upon the base line of the cranial opening due evidently to cerebral bulging. No evidence whatever of necrosis.

## THREE CASES OF TUMOR OF THE BRAIN, WITH AUTOPSY.

BY DR. GEORGE J. PRESTON, OF BALTIMORE.

The first case was that of a boy, aged thirteen years. For a year he had what looked like ordinary epileptic attacks, which were very much lessened by the bromids. Then he suddenly developed diplopia, with some neuroretinitis. Vision was greatly reduced, and there was at first left lateral achromatopsia, and afterward left hemianopsia. Gradually he lost entirely both sight and hearing. The reflexes, both superficial and deep, were lost. He complained of most intense headache, and there was gradual failure of mental power. The autopsy revealed a large tumor, involving the entire right temporal lobe; the occipital lobe was softened and broken down, but the cortical portion of the occipital lobe was not involved. The tumor was a sarcoma. The second case was interesting from the fact that the tumor, a small papilloma, not larger than a partridge egg, burying itself in the right superior occipital convolution, produced intense headache, with double optic neuritis. The third case showed a large sarcomatous tumor involving the temporal lobe, all except the first convolution, the greater part of the occipital lobe, and the lateral lobe of the cerebellum. The symptoms were not very well marked, except headache and mental disturbance. It was interesting to note that the first temporal convolution was not involved, though the rest of the lobe was virtually destroyed, and in accordance with our accepted views concerning the auditory centre there was deafness.



## PRESENTATION OF A BRAIN TUMOR.

BY DR. GEORGE L. WALTON, OF BOSTON.

### ABSTRACT.

The chief interest lay in the question whether operation could have been successfully performed.

The patient, a man of fifty three, seen in consultation with Dr. Phippun, of Salem, was well apparently up to within six months of death, when appeared indisposition, indifference, and lack of ability to figure—symptoms hinted at for, perhaps, six months previously, but not interfering with work. Epileptiform attacks with loss of speech and twitching of right face with curious sensation in throat removed, and toward the end general convulsions. Right hemiplegia and right hemianopsia came on about one month before death, with and double optic neuritis. Pupils were unaffected. There appeared to be numbness of right hand. The superficial reflexes were impaired on the sight. Intelligence was apparently good, the aphasia and agraphia were marked.

Autopsy showed a tumor, apparently glioma, having a round surface, in the angle between Rolandic and Sylvian fissures, two by one and one-half inches, quite sharply defined. Vertical section showed extension inward about one inch with fairly distinct line of demarcation, but no capsule. The tumor extended forward under the healthy cortex to a point beyond the transverse frontal sulcus. Microscopical examination to be reported later.

### DISCUSSION.

Dr. STARR, of New York.—I have a case I would like to add to these of an infiltrating glioma of the left hemisphere in the motor zone in the hand and arm area that occurred last October and was observed carefully by me at my clinic, and was afterwards operated on by Dr. McBurney. The case was in a man without ostensible cause, neither blow or syphilis having preceded the

development of his symptoms. The symptoms had developed gradually in the course of a year, being symptoms first of headache; secondly, gradually increasing optic neuritis, and thirdly, the localized spasms occurring at increasingly frequent intervals, commencing in the right arm and hand. There was, after a time, a slight paresis of the right hand and arm associated with a moderate degree of anæsthesia. No disturbance of the muscular sense. In the last two months before operation the patient developed a mild condition of motor aphasia, talked slowly, sought for words, understood perfectly, but was slow of utterance. Operation was advised. He entered the Roosevelt Hospital and Dr. McBurney performed the operation that he has now performed about sixteen times for me of this semi-circular flap operation. It is altogether the best way to get into the head. It exposes a large area without delay, and in point of time it is much more rapid than the old method of trephining. I have timed the method very carefully, and have never seen the trephine get through in less than fourteen minutes, and I have seen Dr. McBurney in eleven and a half minutes from the time of the first incision get the flap down and the dura opened, which is pretty rapid work considering it is done with gouge and chisel rather than with a saw or in any other way. I mention that incidentally. When the brain was exposed it was perfectly evident that there was an infiltrating glioma and that the glioma was very extensive. It extended backward and upward, no capsule, full of blood-vessels, the merest touch caused immediate hæmorrhage into the substance of the tumor so that little points of blood welled up everywhere. It was evident that it would be practically impossible to extirpate this. The question was considered of taking out a wedge-shaped portion of the tumor, but the bleeding was excessive and it seemed practically impossible to do it without producing fatal hæmorrhage, and, therefore, the wound was simply closed up, and although two or three spots were touched with the cauter to stop hæmorrhage, attempt at ligature failing, the man died in the course of the night undoubtedly from bleeding of the finer vessels all through this tumor. The autopsy showed that the tumor was merely seen upon the cortex in the arm area, but that it actually infiltrated the brain forward and backward causing pretty extensive gliomatous infiltration. This fatal case without success either in the removal of

the tumor or the relief of the symptoms I take occasion to report.

I would say a word with regard to the operation when you are not certain with regard to the diagnosis. I have seen since October last eleven tumors of the brain, and this is the only tumor in which I have ventured to locate and operate. In the other ten I have refused operation excepting in one. That one is of interest. It was a case in which there was no question about a tumor in the corpora quadrigemina. The man suffered intensely from headache which nothing would quiet. It seemed to me possibly justifiable to attempt the operation of Horsley who recommends in case of tumors of the brain where access is not probable to the tumor that it is well to take off a considerable area of the skull in order to give relief to the intracranial pressure, puncture if possible, and reach the fluid through a small puncture from the lateral ventricle. In this case, which was operated on about a month ago, we removed a considerable flap of boggy tissue from the parietal region, but did not succeed in reaching it after puncture in several directions, and simply closed up the wound. The wound healed up perfectly, and the bandages were removed after ten days, but, of course, there has been no relief at all, and within a month the pressure of the dura outward has already produced a swelling the size of a small orange upon the surface of the skull, the soft tissues yielding from the increased pressure from within, and I must say I have concluded from the experience in that case that an operation of that kind merely prolongs the sufferings of the patient and is practically of no use whatever. I should not favor it in any case at all.

The fact brought out by Dr. Fisher's case of no relation between the situation of the tumor and the situation of the pain is a conclusion reached by Vanheim in 1880, and I think that has been confirmed by every collector since that the position of the pain gives no clue to the position of the tumor.

Dr. DERCUM, of Philadelphia.—Some years ago I reported a case in which I had an experience somewhat similar to that of Dr. Fisher. The patient presented intense headache, total blindness and total deafness, and a number of symptoms pointing to tumor, but nothing of localizing value. We thought by adopting Horsley's expedient, we could at least relieve the pain for a time. We trephined, tapped a ventricle and did for a time

relieve the headache, and some of the other symptoms temporarily improved. It is worth while, perhaps, to multiply such experiences. The patient died some days after the operation, but he was comfortable as long as he lived.

Dr. DANA, of New York.—Dr. Fisher and Dr. Starr referred to the method of operation, and I would like to refer to one point in connection with that. I cannot speak very positively in regard to operations by the flap method or with the chisel on adults, but upon children and infants I am sure that it is a very bad method, I mean by that the use of the chisel. I think that with the saw, as it has been now perfected, the operation can be done very much more quickly without causing any concussion of the brain, and with vastly better results. As a good many know, Dr. Powell, of New York, has had his instrument perfected, and has operated now upon, I think, twenty-five cases of idiocy and epilepsy in young children with only two deaths, a mortality which is vastly better than that of any other surgeon. I have seen him operate, and he does it more rapidly than any other operator I have watched. And I think there is no question at all as to the best technique surgically for that class of cases. I speak of it in connection with other cases, because I know in a meeting of the German Surgical Congress recently, Bergmann in his presidential address referred to the electrical saw as among the improvements in modern brain surgery, and so I inferred that German surgeons are using it in adults.

Dr. GEO. W. JACOBY, of New York.—I think I can cast a little light on this question. I witnessed two operations upon the skull in adults by means of this electrical saw, and its use was extremely unsatisfactory. Every now and then it got wedged in between the very narrow rim of bone, then the machine would have to be stopped. The amount of hæmorrhage from the bone was very much greater than usual, and we came to the conclusion that the chisel operation was by far the best. Of course, two cases do not say much. It was a new instrument and we were not familiar with its defects.

As regards the question which Dr. Starr has raised as to trephining with function of the ventricles, simply for the relief of pressure, I should like to say that that is an operation which I have advocated several times; I do not care here to refer to the results obtained in those cases, nor to the practicability of relieving pressure

through puncture of the ventricles, but desire to call attention to an operation which has excited considerable attention in Germany, but which here has received but scant notice; it is that operation known as lumbar puncture, first described by Quincke and recommended by him as a therapeutic agent. Puncture is made between the third and fourth lumbar vertebræ and the cerebro-spinal fluid oozes out through the canula in varying amount. I have given attention to this question for some time, but will at present only say that this little operation is very easily carried out, it not being any more difficult than a thoracentesis, that with the greatest facility from 20 to 40 cc. of fluid can thus be removed and the brain pressure correspondingly decreased, and that in those cases of brain tumor, in which I have thus relieved the pressure, the headache has been temporarily, but in some cases only very slightly diminished.

Dr. DERCUM, of Philadelphia.—I wish to allude to the instrument invented by Dr. Cryer, of Philadelphia, for cutting through the skull, described in a number of the *Medical News* not many months ago. It consists of a burr which is made to revolve many times a second by a dental engine. A small opening is made in the calvarium, the burr is inserted, and cuts a flap of bone in a marvelously short space of time. The point is guarded by a steel button. The hæmorrhage can be controlled with very little difficulty.

Dr. JOSEPH COLLINS, of New York.—An observation I made sometime ago while Dr. Gerster was about to remove a brain tumor for me I had not seen mentioned in the literature, and this anent the remark Dr. Fisher made about the innocuousness of the chisel in opening the skull. The skull was rather a thick one, and it was noticed from the first that after Dr. Gerster began the use of the chisel that the patient did not seem to stand the operation well. That is, he grew very pale, the pulse became weak, the extremities cold, etc. We had been giving him chloroform, and at the suggestion of Dr. Gerster ether was exchanged for that with the hope that the respirations would pick up and the pulse become better, but instead of that the patient kept growing worse. I then held his wrist and very soon noticed that just as soon as the operator would stop hitting with the chisel the pulse would go up to sixty or seventy, that every time he would strike three or four times in rapid succession the pulse would go down to forty or thirty.

five. It was not one single observation, but we kept it up fifteen or twenty minutes. At one time he went so low that we applied artificial respiration. Dr. Gerster went on with the chiseling and the pulse again went down to thirty-five. I can bear testimony to the electrical saw mentioned. In the beginning I did see conditions similar to those described by Dr. Jacoby, but never with the perfected saw. As Dr. Powell operates now, it is exactly similar to a buzz-saw going through a pine plank. He will take a piece of shingle and set the saw to the depth of the shingle and pass it through the shingle as you pass a knife through ice cream.

Dr. STARR, of New York.—I should like to ask if Dr. Powell has done this exclusively on children.

Dr. COLLINS, of New York.—Dr. Hammond or Dr. Dana can answer that better than I. The skull of a patient fourteen or fifteen years of age, on which I saw him operate was quite as thick as the skull of many adults. I have never seen any of the excessive hæmorrhage that Dr. Jacoby has mentioned since the perfection of the saw, but in the beginning I did see it. I feel anxious to bear testimony to the efficacy and worth of this method of operating on the skull, particularly since making the observation with the chisel and mallet which I mentioned, and I should like to ask if that has been noticed by others.

Dr. HAMMOND, of New York.—Dr. Powell has operate on seven cases for me with the electrical saw, six infants and one adult. The infants varied in age from one to seven years, and the adult was twenty-two. As Dr. Jacoby states, when Dr. Powell first began to use the saw it was worked by a battery inadequate for the power required, and the saw would stick in the bone. In the first case Dr. Powell operated on for me, the skull of the child was not unusually thick, and yet the saw was arrested every inch or two. The method then used was crude. But after that the saw was improved upon, the strength of the electrical energy was increased, and the saw as it is now used and in the last few cases worked admirably. Lannilongue's operation was performed upon all the children. The operator began by making a trephine opening in the frontal bone about an inch to one side of the median line and a similar opening in the occipital bone. A thin metallic protector was slipped from one opening to the other and between the dura and the skull. He then ran the saw between one opening

and the other, and then again about a quarter of an inch from and parallel with the first cut. The piece of bone thus severed was then lifted out. Other pieces of bone were also removed, the incisions beginning at the same trephine openings and extending for, perhaps, three inches at right angles to the opening made by the removal of the first strip of bone. It did not take three minutes after the saw once started to take out the piece of bone. The whole operation requires, perhaps, twelve minutes. But in other operations, such as Dr. Abbe performed for me the other day, I do not see how the electrical saw can be utilized, because it is particularly adapted where you want to make straight lines, but where it is necessary to make a curved line I do not see how the electrical saw can be advantageously employed, but on the straight line incision I never have seen any operation performed in less time or with less shock, or in a neater way than that done with Dr. Powell's electrical saw.

Dr. DERGUM, of Philadelphia.—The point Dr. Collins communicated to the Association I noticed in the very first case of extirpation of the Gasserian ganglion Dr. Keene performed. He used the chisel, and every time he gave a series of blows the pulse went to fifty, forty-five, forty, and after a little rest it would come up. I attributed it to inhibition from irritation of the dura.

Dr. FISHER, of New York.—I would simply like to reply in regard to the effect of the chiseling on the circulation, that I have never seen death from that operation on the table, and the after-effects do not indicate that there has been much cerebral shock or concussion. I have not tested the point Dr. Collins referred to where the pulse goes down to that extent. The after effect on the patient certainly has not been bad. The specimen of skull here was not shown as a successful result of that operation, but the result there was due to hernia cerebri preventing proper union going on, but as I said in the paper, I have had cases where at the autopsy the union has been perfectly good in every way.

## A CASE OF INJURY TO THE LEFT ANGULAR GYRUS.

BY C. EUGENE RIGGS, M D.,

St. Paul, Minn.

I wish to present a case of injury to the left angular gyrus which seems to me interesting because of the ocular symptoms it occasioned.

The effect upon the vision of monkeys of the extirpation of the angular gyri and of the occipital lobes respectively, has occasioned great difference of opinion among eminent physiologists and investigators, but it is generally agreed by recent writers upon cerebral localization in man that the visual area of the human brain is located in the occipital lobes, especially involving the cuneus. This conclusion is based upon pathological indications, which alone are trustworthy. Some writers also locate an additional visual area in the angular gyrus, while others among whom, I believe, are counted some of my confreres in this society, reject this belief and hold that the visual field is confined to the occipital lobes.

May 7, 1895, Thomas Rush was sent to me for examination by Dr. B. J. Merrill, of Stillwater. I quote an account of the case previous to the time I saw it, kindly furnished me by Dr. Merrill.

"Thomas Rush, a guard at the Minnesota State Penitentiary, was struck two blows upon the head with a machinist's hammer in the hands of an insane convict. Rush was felled, but lost consciousness for a few seconds only. He came unaided from the shop to the hospital department. He was able to give a clear and accurate account of the incidents preceding and following the injury, as well as the number of blows received. He presented two scalp wounds adjacent to each other and located to the left of the vertex of the skull and just above the junction of the left parietal bone with the occipital. Examination revealed a depressed fracture a little over an inch in length and one-eighth inch in depth. Shock was not present. Not a symptom indicating injury



to the brain was present. It was, therefore, deemed advisable not to operate at once, but to dress the wound, keep him under observation and await the incidents of the coming few days. Rest in bed, salines, light diet and ice to the head was ordered. He remained in the hospital about ten days and then was allowed to go to his home. During this time his recovery was uninterrupted by any untoward symptom. There was not a vestige of headache, no disturbance of the motor or sensory apparatus, nor the least interference with intellection. There was an absolute absence of symptoms either of compression or concussion. He became very anxious to return to his work and was allowed to do so about four weeks subsequent to his injury. About six weeks subsequent to the injury he commenced to complain of headache and a sense of confusion while on duty in his shop where there was considerable noise. This condition grew worse, and he was finally obliged to give up his duties again and go home—a period of eight weeks after the injury. Nothing relieved the headache. He was able to be about and walked from his home to my office, nearly a mile, to consult me every two or three days. Finally, about the first of May, he complained of trouble with his vision, though nothing abnormal could be discovered concerning the mechanical apparatus of the same."

When Rush came to me for examination he said he slept well, but suffered from an irritability of temper unnatural to him and had perceived that his memory was not as good as formerly. On examination the motor and sensory reflexes were all found normal, the interscapular not being present on right side. The visual field, accommodation response of the pupil to light of both eyes was normal, and the vision of the right eye was perfect. Vision in the left, being the same side as the injury to the head, was very defective. At four feet he could see the outlines of persons, but could not distinguish one from another, nor a man from a woman, except by the outline of garments. At nineteen inches he could see nothing on a printed page; at seventeen inches, a blur; at ten inches could see lines; at five and one-half inches could see the letters, but said, "They do not convey anything at all to me. They might be Russian for all they mean to me." This remark naturally suggested word-blindness, but on bringing the book still closer he read a line, then stopped, saying he could not read any more.

That the visual trouble was not hysterical was demonstrated by a careful physical examination for the elimination of that possibility. There was no loss of color sense and not the slightest evidence of any of the stigmata of hysterical state. I used Flee's box, more as a matter of curiosity than anything else. He appreciated the presence of the two colored disks. The defective eye was shown to be as he described it, for the disk which should be perceived by it was recognized, but more dimly.

It was decided to trephine. I saw him again before the operation. Vision with the left eye had become even more difficult, and while the vision of the right eye was unaffected, it seemed weakened. He could not read more than five minutes without tiring it.

Trephining was performed by Dr. Merrill, assisted by Drs. Clark, Boleyn and Ball, May 12, in the presence of Drs. Crafts, Baker and myself. Two buttons were removed with trephines, one an inch and a half in diameter, the other an inch in diameter, the entire opening in the bone being slightly more than two inches the longest way. The inner margin of the opening came almost to the median line and its lower border touched the parieto-occipital suture. A small nipple-shaped spicula of bone was found at the point of juncture of the trephine openings, and the dura was discolored beneath and just posterior to the main depression. At the end of the operation the patient was in a critical condition, refusing to breathe and the radial pulse absent. Measures were taken to resuscitate, and after half an hour the patient rallied and was put to bed.

The vision of the affected eye was tested after the operation by Drs. Boleyn and Ball and was found to be greatly improved. The patient could recognize and distinguish between them when some six feet away and could also count the fingers on a hand held up at the foot of the bed.

About five hours after the operation the patient died suddenly. A post-mortem was held the next morning. There was found general adhesion of dura to skull; infiltration and adhesion of dura to pia about one inch in length at the upper extremity of the fissure of Rolando, and scattering adhesions along the whole length (anteriorly) of longitudinal sinus; injection of pia generally; softening of brain cortex at point of strongest adhesions; softening of greater portion of angular gyrus

and of region between the angular gyrus (using the term in its most limited sense) and supra-marginal convolution; softening of anterior portion of gyrus fornicatus.

The occipital lobe was carefully examined by Dr. Crafts and myself, and no lesion was found present there.

This case seems to me noteworthy for several reasons. First, because of the absolute demonstration it gives that the depression over the angular gyrus was the cause of the dimness of sight, which was so remarkably improved after the operation. Second, because of the fact that the visual disturbance was homonymous instead of being a crossed amblyopia, which is, according to Gowers, the only recorded visual symptom resulting from lesion of the angular gyrus. Third, because of the remarkable absence of symptoms of the subacute meningitis discovered by the autopsy.

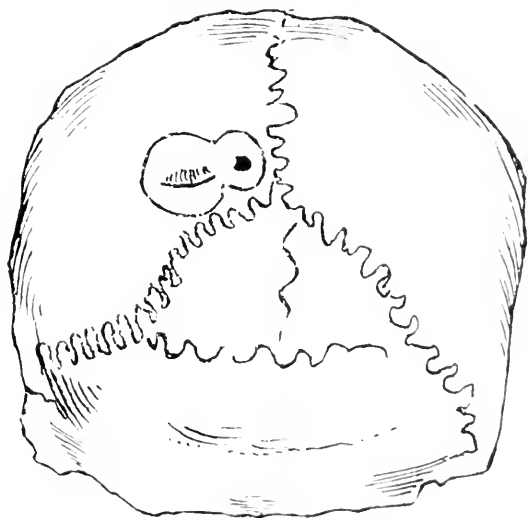


FIG. 1.—Seat of lesion in the superior posterior angle of the left parietal bone. A crescentic depression in the bone about one and one-eighth inches long, five eighths wide and one-eighth deep, the convexity pointing downward, another circular, very slight depression between that and the sagittal suture. Two buttons removed with trephines—one an inch and a half in diameter, the other one inch in diameter, the entire opening in the bone being slightly over two inches the longest way. The inner margin of the opening coming almost to the median line and its lower border touching the parieto-occipital suture. A small nipple-shaped spicula of bone at the point of juncture of the trephine openings was found and the dura was decidedly discolored beneath and just posterior to the main depression (probably just over the softened cortical area).



FIG. 2.—Side view of skull with left parietal removed showing the brain.

1. Fissure of Sylvius.
2. Supra-marginal convolution.
3. Approximate location of area of softening.

#### DISCUSSION.

Dr. STARR, of New York.—I should like to ask whether there was a history of defective vision in that eye before injury, and whether the eye was examined for refractive error.

Dr. RIGGS, of St. Paul.—The patient stated that the eye was perfect before injury.

Dr. STARR.—Was his vision improved by glasses at all?

Dr. RIGGS.—I did not try them.

THE PATHOLOGY OF HEREDITARY CHOREA.  
REPORT OF A CASE WITH AUTOPSY.  
RECORD OF ANOMALIES IN A DEGENERATE BRAIN.

BY CHARLES L. DANA, M.D.

THE clinical history of hereditary or Huntington's chorea is very thoroughly known, and I should not feel justified in reporting a case on the ground of its symptomological features alone. In the present instance, however, the patient died somewhat early in the disease, from typhoid fever, and I was able to make an examination of the brain before the malady had advanced to its last stage. The patient had also a most remarkable hereditary history.

CLINICAL HISTORY.—John C. was thirty-seven years old at the time of his death. He was single, born in New Jersey, and was sent to my clinic at the Post-Graduate School in 1892 by Dr. George R. Elliott, to whom I am greatly indebted for notes in the case and for assistance in preparing the pathological specimens.

The hereditary history of the patient is extremely interesting and almost unique in the extent and definiteness of the hereditary transmission. Through correspondence and personal interviews I have been able to trace back his disease into the fifth generation. The accompanying table will show better than any elaborate description exactly how the disease has been transmitted.

The original sufferer was a great great-grandmother, who was born in Scotland and who died, as tradition gives it in the family, of chorea. The great-grandmother was also born in Scotland and died in New Jersey of choreic dementia. The grandmother also died of a chorea which began after the age of thirty. The mother of the present patient also died of chronic chorea, which developed after the age of thirty. She died at the age of forty. The patient himself began to develop choreic

# GENEALOGY OF A CASE OF HEREDITARY CHOREA.\*

•

*Mother, b. Scotland; d. of chora.*

11.

Mrs. H., b. Scotland; d., New Jersey, of *chorea*.

110

Mrs. P. Mrs. Van B.	Sister,	Sister,	Brother, healthy;	Brother, healthy;
	healthy, healthy,	d. at sea.	d. at sea.	d. at sea.

15.

*Eller-Sister, Younger-Sister, Mrs. C., Younger-Brother,*  
d. 46; began, 30. d. 40. 20-43. 25-40. Daughter, m.; d. of heart disease, 45.

5

v. Son, d. of Son, 20. *Johu*, Daughter, Daughter, m., Three children,  
gastritis, 23. well. *patient* 36. m. 33. 23; phthisis. 24; well. healthy.

Two children.

\* The persons in italics were those who had chorea.

symptoms at the age of about thirty-three and died at the age of thirty-seven from typhoid fever, having reached about the middle stage apparently of his disease, for, judging from the course of it in his ancestors, the malady lasted ten or twelve years. The family history shows also hereditary chorea in a great aunt, and in the aunts and uncles of the present patient. None of the brothers and sisters had yet begun to suffer from the affection, but the patient was the oldest, and it still remains to be seen whether the disease will be developed in the other members of the last generation.

The genealogical record shows that the disease is transmitted entirely through the female members of the family, but that all the female members of the family did not transmit it, even though some of them had the disease themselves. It also shows that the disease developed with almost absolute definiteness in time at the age of thirty-two or thirty-three years. So far as the accounts go, the symptoms seem to have been pretty nearly the same, the patients developing at first choreic movements of marked type, and then mental symptoms which ended in chronic mania and dementia. The disease was in the earliest manifestations more of the nature of the ordinary chorea, but, as it became transmitted from generation to generation, the motor element became less marked and the symptoms of a progressive dementia were the most prominent.

The disease began in my patient with no exciting cause, and the first symptoms were a slight disturbance in articulation. This was accompanied with twitching of the lips and choreic movements of the facial muscles. As the disease advanced, twitching movements of the head and of the arms became noticeable. The gait was of the peculiar loping or high-stepping character. A short time after the development of the speech troubles the patient showed some disturbance of the mind. He became easily excited, and sometimes was violently angry over slight things. His memory failed, and he was unable to perform the ordinary duties of his work, which was that of a pedlar. His father finally consented to his admission to the hospital, as he was of no help at home, and his temper was becoming a somewhat disturbing element in the family.

A physical examination of the patient on admission showed him to be a strongly built, muscular man of about 5 feet 7 in. in height. His gait was characteristic of

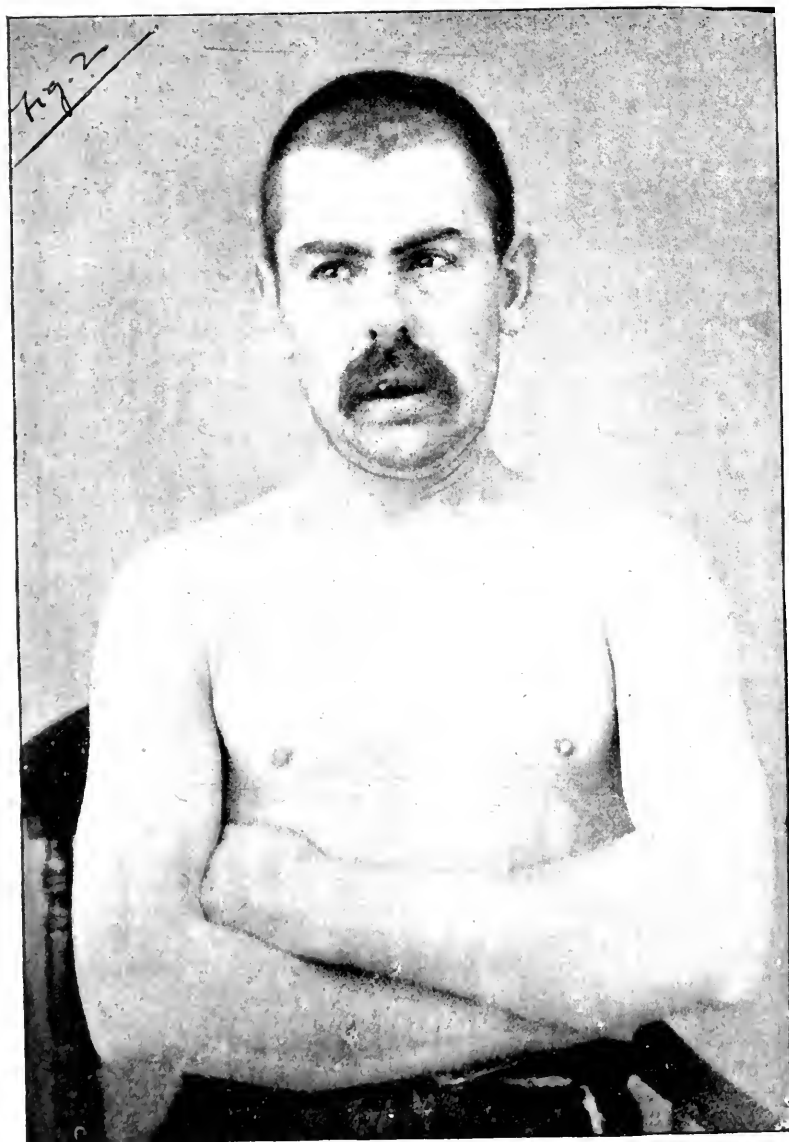


FIG. 1. Physiognomy of patient in fifth year of the disease.



the hereditary chorea, that is to say, his steps were long and high, and his body swayed to and fro in walking; his head at the same time made a nodding motion so that his appearance was very striking. He showed some twitching movements in the facial muscles and occasionally in the shoulders and arms. The tongue protruded with some difficulty, and he did not seem to have full control of it. There was no nystagmus, but the eye muscles would twitch at times. He had no paralysies or atrophies, no pains, or anæsthesias, and no trophic disturbances of the skin. He could stand with the eyes closed and showed in no way any evidence of ataxia; the knee-jerks were normal. His bodily organs—lungs, heart, kidneys and digestive tract were all healthy. His mind was in a weakened state; his memory was defective; he was slow in comprehension, irritable in temper and flew out in the most violent rage at times. He was, however, not yet in any great degree silly or demented, and was able to answer questions quite intelligently. He was fond of being left alone and would sit in his chair reading Shakespeare's plays or Gibbon's History of Rome, apparently enjoying them. Later he took to newspapers and betrayed his intellectual decay by giving up the classics for the daily news.

He continued in the hospital most of the time up to July, 1894, a period of about two years. During this period his motor symptoms did not change very much. He still had the peculiar high-stepping gait, nodding movements of the head, the choreic twitchings of the face, the peculiarities of speech. His temper became somewhat milder and he himself became rather weaker in mind, though still able to do ordinary work about the wards.

In January, 1893, I made a trephine opening over the motor centre for the head, neck and eyes on the right side of the brain. This was done with the consent of his father and himself, and the operation was performed in the hope that by some mechanical interference of this sort the natural progressive tendency of the disease might be checked, as had been reported to be the case in general paresis. As a matter of fact this result did follow, and for five or six months after the operation he was decidedly brighter and better. He then gradually relapsed into his former state. In June, 1894, he developed typhoid fever, the fever running from June 17th to July 13th, when he died. The symptoms during this



FIG. 2 Showing location of trephine opening.

time were those of a somewhat severe type. The duration of the high temperature was from July 3 to 14, about eleven days, so that he may be said to have died in the second week of the disease. Death was caused by intestinal perforation and septic peritonitis.

THE POST-MORTEM was made by my house physician, Dr. Pearce, to whom I am indebted for the notes, and I have also to express my indebtedness to my colleague, Dr. Alexander Lambert, through whose courtesy I was able to obtain the specimens.

The autopsy showed the usual changes in the intestines that characterize typhoid. There was a distinct point of perforation in the ileum. The kidneys were

small with numerous cystic tumors over the surface, but not otherwise diseased. There were nothing abnormal about the lungs, liver or spleen, except such changes as were due to the fever. The heart was small; the wall of the right ventricle thin; the heart muscle pale; the valves were normal and there was no endocarditis.

On removing the calvarium the dura mater was found to be thickened and adherent to the bone along the longitudinal fissure, and also firmly adherent at the seat of the trephine. It was not, however, adherent to the arachnoid beneath, nor was there much thickening of the pia-arachnoid. The brain itself was congested, but showed no other marked changes to the naked eye, the vessels not appearing diseased. There was not any great amount of œdema. The brain and spinal cord were placed in Müller's fluid, hardened and then subjected to examination by various methods, with Weigert's stain, carminate of soda, logwood, and methyl blue. Owing to my absence at the time of the patient's death it was not possible to make any stains by the method of Nissl or that of Lewis.

ANATOMICAL ANOMALIES OF THE BRAIN.—The study of the present case from its pathological aspect presents two features of striking interest. One of them is that of the gross character of the brain as compared with normal brains; the other, that of the microscopic findings and the minute changes underlying the course of the disease. It seemed to me that a brain belonging to a family having such a striking history of transmitted degeneration might, perhaps, present some peculiar characters. Here was a man whose ancestors for four generations had with the persistence and certainty of a machine begun to go through precisely the same disease changes soon after the age of thirty, although apparently sound and vigorous men and women before that. There was evidently at their birth some potentiality for early degeneration. Whether the brain itself showed on the surface characters which would indicate that it was an organ that would be liable to early deterioration was a question which I sought to answer.

I first of all made some examination of the head and skull. This gave, however, rather few of the stigmata of degeneration. As the picture shows (Fig. 1), the face is not that of a very intelligent man, but the head is normal in size ( $gc = 54$ .) and symmetrical. The patient had coarse hair, thick bushy eyebrows, badly set

teeth, a high, narrow, arched palate, and a slight amount of facial asymmetry. He was a man of strong muscular development. The brain was carefully photographed after it had been hardened in Müller's fluid for three months, and the pia mater removed. I made some careful studies then of the arrangement of the convolutions, taking measurements of the different parts. I also made a series of one hundred and fifty measurements of the thickness of the different parts of the cortex. The results of these studies I append here.

#### THE FISSURAL ARRANGEMENT.

##### 1. *The Fissure of Rolando.*

*Left.*—The fissure measures 12 ctm. in its total length measured with a moist thread. Above it does not quite reach the longitudinal fissure. At the lower third it is interrupted by a bridging convolution and the lowest third runs into the fissure of Sylvius.



a.

b.

FIG. 3. Brain of hereditary chorea. *b*, left hemisphere, interrupted fissure Rolando.

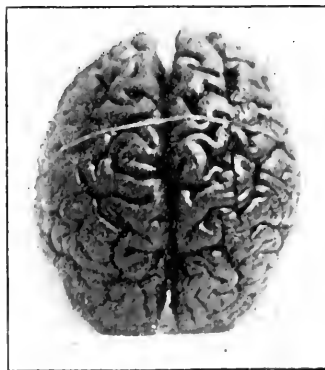


FIG. 4. Showing absence of *pli de passage* on right side.



FIG. 5.

*Right.*—The fissure above runs over into the longitudinal fissure. It is  $12\frac{1}{2}$  ctm. long and forms a normal angle with the longitudinal fissure. It presents no special peculiarities.

2. *The Fissure of Sylvius.*

*Left.*—The posterior branch is quite long, measuring 7 ctm. and ends in a fissure running at right angles to its course which is confluent with the first temporal below. The fissure is itself confluent with the fissure of Rolando. Vertical branch is very long, measuring 4.2 ctm. The fossa is covered; the anterior branch is of average length.

*Right.*—The fissure of Sylvius is long and straight. The posterior branch measuring 8 ctm. The retro-centre fissure runs into it. The anterior and vertical portions are very long, the frontal branch measuring 4 ctm.; the vertical 3.7.

3. *The Parietal Fissure*

*Left.*—The superior part of the retro-centre fissure is irregular in course and runs into the fissure of Rolando. The inferior part is continuous with the middle part of the parietal, and the middle part of the parietal is entirely independent of the occipital.

On the *right* hemisphere the superior and inferior parts of the retro-central fissure unite and form one continuous fissure parallel to the fissure of Rolando. The parietal part is connected with this, but is entirely disconnected with the occipital. This entire separation of the parietal part from the occipital portion on both sides is the only peculiarity of the fissure. I have seen it occur in a low type negress' brain, but I cannot say that it might not be found in normal brains.

4. *The Occipital fissure*, left side, is well marked and presents nothing abnormal. The external branch measures 1.8 ctm. in length. On the right side the occipital fissure is deep, very well marked and runs into the par-occipital of Wilder, so that there is no superior external *pli de passage*. This latter is the only peculiarity that I note in connection with the occipital fissures. The anterior occipital fissure of Wernecke is well marked in both right and left hemisphere. The anterior occipital as described by Eberstaller is very poorly marked.

The first temporal on the left side is short, runs into the fissure of Sylvius and also into the second temporal, and does not run into the anterior occipital of Wernecke. On the right side the first temporal is broken in two parts, the second being confluent with the second temporal.

The third temporal is broken in two parts on the left side and in three parts on the right.

The fourth temporal is a continuous fissure ending in a T fissure. In the right hemisphere from the under surface of the occipital lobe is a transverse fissure, making the head of a T with the fourth temporal fissure and running from the junction of the occipital and calcarine fissure to the external border of the occipital lobe.

The inferior præ-central fissure is normal and confluent with the second frontal fissure. The superior part of the præ-central fissure is also well marked and continuous, not confluent. The medi-central fissure is distinct and not confluent, the frontal convolutions have, therefore, a rather simple type. All this applies to the left hemisphere. On the right side the inferior præ-central is long and runs into the second frontal fissure, which is short with many transverse fissures. The superior part of the præ-central is distinct and continuous, and so also is the medi-frontal fissure, which is not connected with either the first or second frontal.

The calloso-marginal fissure on the right side is continuous. On the right side also continuous and not confluent upon either side.

SUMMARY OF ANOMALIES. In reviewing these observations upon the arrangement of the fissures, I note a few peculiarities which may be considered of significance. These are the interruption in the fissure of Rolando by a bridging convolution. This anomaly is certainly rare, and probably has some significance as an

evidence of imperfect evolution of the brain. The arrangement of the retro central fissure so that it parallels without any interruption the fissure of Rolando, and runs into the fissure of Sylvius is perhaps of some significance also. The confluence of the occipital fissure with the par-occipital, thus cutting off the *pli de passage* is a decided anomaly. The simple arrangement of the frontal convolutions with the fissures well divided and not confluent, and the peculiarities of the anterior occipital of Wernicke and of the fourth temporal indicated in the description above, complete the list of what may be called anomalies. Taken as a whole, it will be seen that the brain carries in its configuration two or three striking evidences of its being an peculiarly developed organ.

I append now some measurements of the different parts of the brain which may be of use for comparison in other studies.

## MEASUREMENTS IN CENTIMETRES.

Weight, 49 oz.

	Right.	Normal average.	Left.
Trigonum to occipital pole . . . . .	34.	33.6	33.5
"    "    fissure . . . . .	28.	28.	
"    fissure Rolando . . . . .	21.5	22.	22.5
Total length of brain . . . . .	17.5	16.2 to 17.2	
"    "    corpus callosum . . . . .	7.1		
Occipital fissure to occipital pole . . .	6.		5.
Fissure of Rolando,			
Sinuous length . . . . .	12.5	11.3	12.
Straight " . . . . .	11.		
Fissure Sylvius,			
Posterior branch . . . . .	8.	7.28	7.
Fossa . . . . .	2.5		2.
Frontal branch . . . . .	4.		
Vertical " . . . . .	3.7		4.2
Occipital Fissure external branch . . .	2.		1.8
Central Convolutions,			
Breadth of both lower parts . . .	3.		
Pre-central, average of 10 measure ments . . . . .	1.		
do. of post central . . . . .	1.2		

THE RELATIVE CORTICAL THICKNESS.—A good deal may be learned regarding the condition and development, or lack of development, of the brain by studying the thickness of the gray matter in the different areas.

I pursued this plan with the brain of a Peruvian Indian, and I have examined other brains in similar

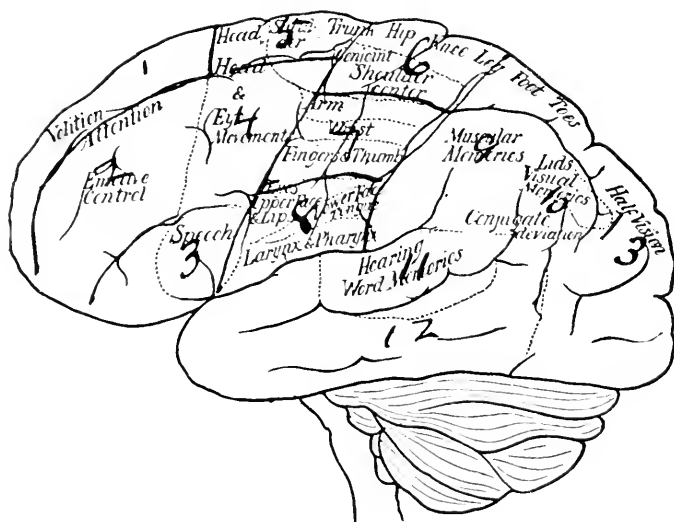


FIG. 6.

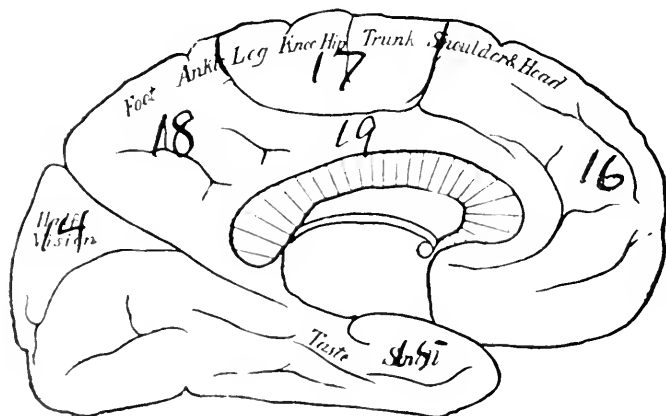


FIG. 7.

Figs. 6 and 7, showing the 19 sub-divisions of cortex for purposes of measuring thickness.



fashion. In order to see whether this method would throw any light upon the morphological characteristics of the present case, I made a series of one hundred and fifty measurements of the gray matter. These were made largely upon the cortex of the right hemisphere, but in the more important parts, viz., the central convolutions, the frontal and occipital lobes, examinations were also made of the left hemisphere. Measurements were made only of the convexity of the convolutions. I believe this to be the best practical way of making the examination. I have not found that the method described by Donaldson of measuring the cortex and the two sides of each convolution presents any great advantages. I took the brain after it had been hardened for three months in Müller's fluid so as to bring out the



FIG. 8. Diagram showing the thickness of the cortex in the normal brain in hereditary chorea. Dotted line indicates normal curve, continuous line that of choreic brain.

coloring of the cortex well. I then divided it into nineteen different parts in the way shown on the diagram. (Figs. 6 and 7). With a Valentine knife sections were made from these different areas, they were measured and averages of the thickness of the convolutions for the different parts were computed. I then compared these with the measurements of normal brains as given by Donaldson, and prepared a table which shows the relations between the thickness of the cortex in my case and that of the normal brain. It will be seen (Fig. 8) that the line in my case runs a little below that of the normal for every part except the hippocampus and the occipital fissure, but that the relatively lowest part is in the præ-frontal, the speech and the sensory motor centres. In what may be called the latent areas like the gyrus fornicatus, the temporal lobes and in the occipital lobes there is not very much falling away from the normal.'

#### MEASUREMENTS OF THE THICKNESS OF THE CORTEX.

The following are the averages of 150 measurements, in millimeters:

Prae-frontal,	2.85 (20 measurements).
Third left frontal,	2.57 (left same as right).
Superior central,	2.2.
Parietal lobule,	2.2 (23 measurements).
Third central,	1.9 (9 measurements).
Lower central,	2.2 (9 measurements).
Hippocampus,	3.8 (15 measurements).
Gyrus fornicatus,	2.7.
Insula,	3.8.
Temporal, <sup>1</sup>	3.2 (13 measurements).
Temporal, <sup>2</sup>	3.1.
Occipital,	2.1 (14 measurements, left same as right).

#### MICROSCOPICAL EXAMINATION.

The microscopic examination of the brain was made by me without having previously read the article of Oppenheim; as I did not wish to study the sections with any preconceived ideas as to what I should expect. I examined sections of the central convolutions from both sides, also the parietal, occipital and frontal convolutions. The stains that were made with carminate of soda and then stained a short time with logwood gave the best pictures. I noted that in some sections very few changes could be seen. The membranes and the blood-vessels seemed to be fairly normal. I did not find

abnormal changes in any part of the brain except in the central and to a less extent in the frontal convolution, the changes were most marked in the central convolutions. These showed a decided thinning of the gray matter, the pia mater was slightly thickened, but there was no meningitis strictly speaking. The tissue was in some sections very vascular and injected. The walls of the blood-vessels were slightly thickened, but not to any great extent. The perivascular spaces were not very much enlarged, nor were there any cells to be seen deposited around the walls of the blood-vessels. The nerve cells showed the most striking changes, especially those in the second and third layer, including the small and pyramidal cells. These cells did not stain with carmine very well. The first layer is much affected, being in places decidedly thin, so that the second and third cell layers are brought close to the periphery. The cells of the third and fourth layers are shrunken. The processes in many instances cannot be seen; the bodies look granula; their nucleus is enlarged. One in some cases sees small bits of pigment. In some of the areas that are most affected the small cells of the second and third layers have apparently been entirely destroyed, leaving small spaces and giving to the section a reticulated appearance. In the affected parts one may see small cells, probably scavenger cells, lying around the pyramidal cells. Nearly every one of the nerve cells in certain areas has from one to three of these small scavenger cells in the pericellular space fastened apparently upon the body of the nerve cell. This, perhaps, is chiefly marked in the third layer. The layer of the large pyramidal cells (fourth layer) is less affected, but some of these cells are nearly round in shape and have lost their processes and are evidently badly degenerated. The blood-vessels, as I have stated, in the affected areas are not very much changed, though the walls are somewhat thickened. Occasionally a high degree of vascularity of the parts occurs which may be due to the fact that the patient had had typhoid fever and in estimating the changes in the body of the cells and in the blood-vessels, I have taken this fact into consideration. These serious degenerative changes in the cells, however, are evidently of long standing and could not have been due to any febrile process, because they only occur in certain parts of the brain. A special examination was made of the third left frontal convolution, owing to the fact

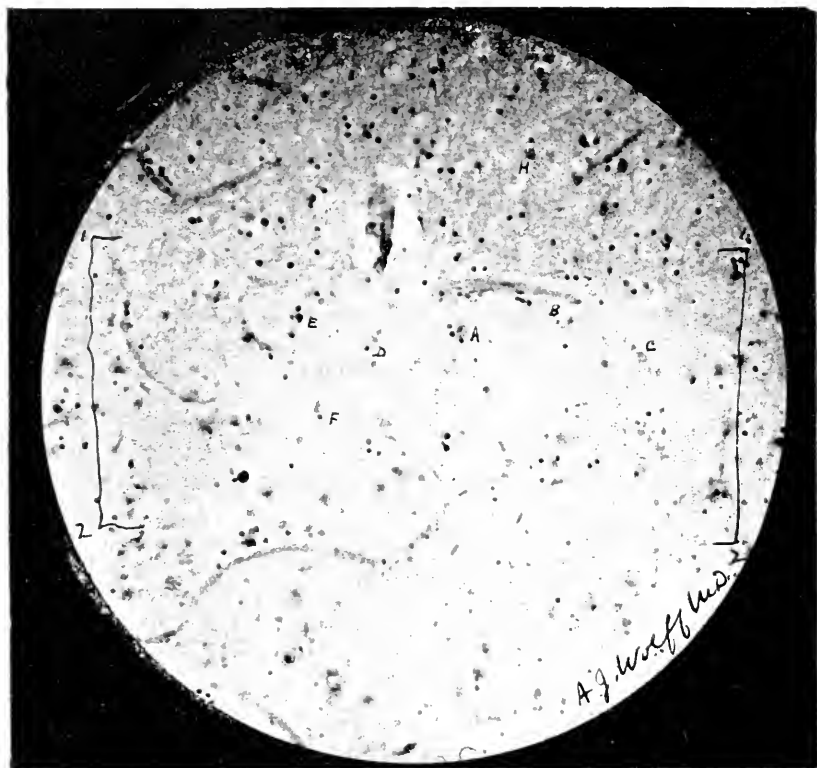


FIG. 9. Photograph of mid-central convolution  $2_3$  in. objective.

that the patient had disturbances of speech. But I could not discover any very characteristic change here. The sections of the occipital lobe showed no peculiar changes. The changes in the second, the middle and the upper part of the central convolutions were the most marked. Sections were made of the spinal cord at different levels, but there was absolutely nothing abnormal in this organ, unless it be that in the upper part there was a slight increase in the connective tissue forming the septa.

SUMMARY OF MICROSCOPICAL EXAMINATION.—Summing up now, the microscopic examination shows that the brunt of the process falls upon the central

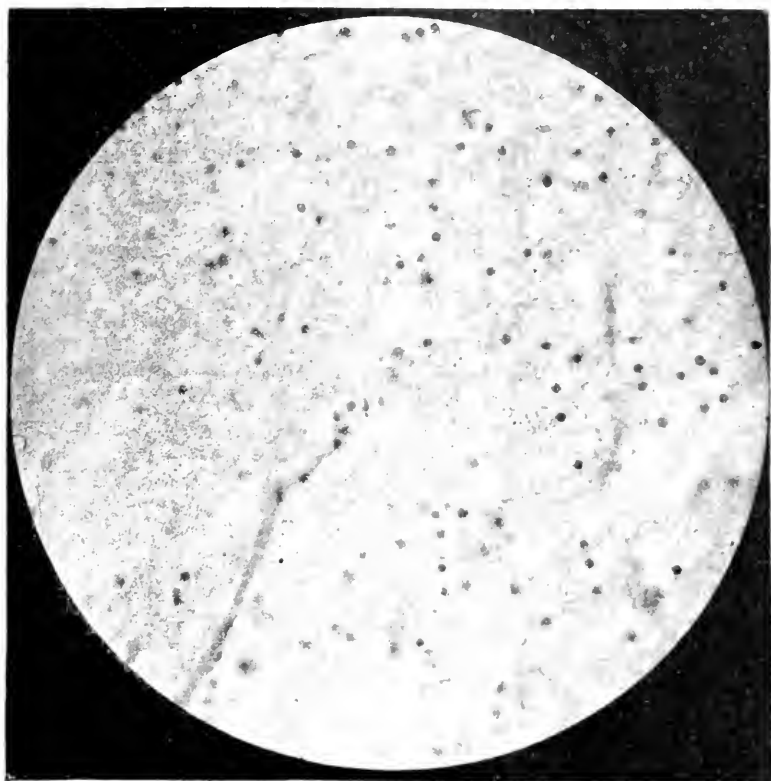


FIG. 10. Showing spaces left by atrophied cells. Same field as Fig. 9.

convolutions, that these are probably the first that begin to suffer, and that they suffer most intensely. It also shows that the process is not evenly distributed, but occurs in patches throughout the affected parts. This would explain, probably, the motor symptoms, the twitchings and choreic movements. This corresponds also with the observations of Oppenheim, that the process in hereditary chorea is a disseminated one. As to the intimate nature of the process I see no results to justify the opinion that it is in any sense an inflammation. There is in my sections no proliferation of connective tissue cells, and no exudation, no accumulation

of leucocytes about the blood vessels, and only a slight amount about the nerve cells. The process in every respect corresponds to one of decay and degeneration; the nerve cell itself is the thing that first begins to die. In this respect hereditary chorea differs, I believe, absolutely from ordinary Sydenham chorea, where the process evidently begins in the blood, and is due to an irritant substance which is poured out from the blood upon the nerve cells in a way described by Sydenham himself. In Osler's work on chorea he concludes his study of the pathology of hereditary chorea with the statement that nothing has yet been found to explain the true nature of the disease. This statement is hardly true at the present time; to be sure, we may never get very much farther in the anatomical knowledge of disease, or if we do succeed in finding more recondite and distinctive changes in the cells, neuroglia and blood vessels, they will not help us to solve the mystery. But we have learned enough to see that the disease is one of decay and death, attacking the cortex of the brain primarily, and affecting one region after another until the part is so destroyed that dementia occurs and finally death ensues. From these facts I think we can, with the help of our knowledge of heredity and of the laws of pathology, deduce a fairly satisfactory explanation of the nature of hereditary chorea. I venture the opinion that the disease is itself a late development of a teratological defect. The patient is born with a brain which is abnormal. The cells originally bestowed upon him were cells that had only the capacity for living thirty or forty years instead of seventy or eighty. Just as cases of hereditary ataxia are born with defective vitality of sensory neurons in the spinal cord, so the cells of the motor cortex are in these people endowed with a short life, and after the strain of thirty years of work they begin to decay. There is no microbe, no poison, nothing in the environment of the individual or in his general physical make-up that produces this. It is simply a death at the age of thirty of cells which should and do in ordinary people live to the age of seventy. With this view of hereditary chorea as being simply a congenital defect, to be classed, perhaps, with idiocy and due to lack of development of the cortex, I believe we have an explanation of the disease which is reasonably satisfactory.

GENERAL SUMMARY.—Hereditary chorea, transmitted

to the fifth generation through the female, death from typhoid fever in the fifth year of the disease. Peculiar anomalies of the brain, interrupted fissure of Rolando, absence of superior *pli de passage*, etc.; general thinning of the gray matter strikingly marked in the central convolutions; areas of great cell degeneration affecting most the angular and small pyramidal layer, the cell defect, primary, the vascular changes minor and secondary. The disease belongs to teratology. It is an innate defect in cell structure.

## DISCUSSION.

Dr. MILLS, of Philadelphia.—Was any anæsthesia present in this case, or any form of sensory disturbances.

Dr. DANA.—There was a condition of general hebetude.

Dr. MILLS.—This case is important in many respects, certainly most important in one. Dr. Dana expresses the true position not only of this case, but of a large number of cases which should be brought together in one class under a common designation, the basis of which designation would be the remark he made that these cases were teratological, and that they simply represented the fact that the individual was born with a certain potentiality of development, that at this age or that he was bound to be prematurely senile. This idea gives a broad basis for classifying certain diseases, such as hereditary chorea and other so-called family forms of disease.

## SPECIMENS OF SPINAL CORD FROM A CASE OF HEREDITARY ATAXIA.

Dr. DANA.—In justice to Dr. Rook, of Quincy, Ills., I would like to pass around some sections of the cord that he sent to me, from a case of Friedreich's ataxia. The man had the disease about seven years, was of a family of four who suffered from this disease. He died of the disease, and this represents the terminal stage of it. I have two photographs which show not only the combined sclerosis which is characteristic of all cases, but some very interesting and peculiar perforations which ran through the whole cord and which I have never seen in any other cord. The holes are due to empty blood spaces, I think.

(*The Proceedings of the Twenty-first Annual Meeting of the American Neurological Association will be continued in the October Number.*)

## Asylum Notes.

### *The Present Standing of Training Schools for Nurses in Hospitals for the Insane.*

	LOCATION.	Date of Organization.	Graduates.		Date Commencement, and Remarks.
			Men.	Women.	
1	McLean Hospital, Mass. . . . .	1882	6	20	No commencement this year on account of moving.
2	Buffalo State Hospital, N. Y. . .	1884	4	8	May 31, 1895 Ninth class.
3	Essex Co. Asylum, Newark, N.J.	1886	7	4	June 7.
4	Kankakee, Illinois . . . . .				Reorganized 1894. No commencement this year.
5	Willard State Hospital, N. Y. . .	1887	3	4	July 1.
6	Kingston, Ontario. . . . .	1888			No commencement.
7	Middletown State Hospital, N.Y.	1888			July 23, 1895.
8	Danvers Hospital, Mass. . . . .	1889	0	4	June 10.
9	St. Peter's State Hospital, Minn.	1889			June.
10	Rochester State Hospital, Minn.	1889	9	7	June 4.
11	Westboro Asylum, Mass. . . . .	1889			January, 1895.
12	Independence Hospital, Ia . . . .	1889	6	13	April 18.
13	N. H. Asylum, Concord, N. H.	1889	0	10	June 11.
14	Utica State Hospital, N. Y. . . .	1890			No commencement.
15	Rochester State Hospital, N. Y.				No report.
16	Eastern Michigan Hospital, Pontiac . . . . .	1890	12	7	March 27, 1895.
17	St. Lawrence State Hospital, Ogdensburg. . . . .	1891			Only Schedule of Lectures received.
18	Michigan Asylum, Kalamazoo.	1891	0	13	
19	Cleveland Asylum, Ohio. . . . .	1891	5	9	
20	Toronto Asylum, Ontario . . . .	1891			No lectures last year; cause, illness.
21	Danville Hospital, Penn. . . . .	1891	9	0	July 12, 1895.
22	Retreat for Insane, Hartford, Ct.	1892			No diplomas given.
23	Western Pa. Hospital, Dixmont				No report.
24	Asylum, Hamilton, Ontario . . .				No report.
25	State Hospital, Warren, Pa. . . .				No lectures last year.
26	Southern Hosp., Evansville, Ind.	1893	5	5	June 6.
27	Friends' Asylum, Frankford, Pa.	1894			
28	Morris Plains Hospital, N. J. . . .	1894			
29	Columbus, Ohio . . . . .				No diplomas.
30	Columbia, S. C. . . . .	1891			No report received.
31	Indianapolis . . . . .	1894			No details.
32	Fergus Falls State Hosp., Minn.	1894			No details.
33	King's Co. Asylums, N. Y. . . . .	1894			No details.
34	Nova Scotia Asylum, Halifax . .		0	4	No commencement.
35	Poughkeepsie State Hosp., N.Y.				No commencement.
36	Binghamton State Hosp., N.Y. . .	1894			No commencement.



In addition to the above, Yankton, South Dakota; Athens, Ohio, and Clarinda, Iowa, announce in their reports their wish to start schools as soon as able. It is also quite possible that others are so working, of whom we have not information. The school movement was from the first so logically beneficial, that it has not received any active opposition, and we have not a doubt that it will soon invade every hospital of this class in America. The reserve now left is rather that of an inertia in form than any other. One man states that he will start a school next season if not removed for political reasons. At least seven states, representing twenty-two hospitals, have had political disturbances during the past five years.

On all hands, however, there is an unqualified commendation of the training school work, and it is recognized as a long step upward. Until drug therapeutics are more certain, it and its collateral work must be recognized as a therapeutical advance of the most important kind. One superintendent writes of it as enlivening new life into the work, and shaking off many fungoid growths. It is safe to say that hospital officers are generally quite appreciative of the work, and that as one local impediment after another of each especial place is removed, nursing schools will come in.

Commencement exercises and any display is deprecated by Dr. G. Alden Blumer (see *Utica report*, 1893), and by Dr. C. K. Clark, Kingston, Ontario; who dislike the "sensational" features and wish to carry the work on "quietly and in a dignified way." Details of their work are, therefore, not known. The Hartford Retreat, the Pennsylvania Hospital at Warren, Hospital at Columbus, Ohio, have lectures without diplomas, for various reasons, not designated. In five of the programmes noted, Cleveland, Ohio; Evansville, Ind.; Willard, N. Y.; Independence, Iowa, and Rochester, Minn., nurses were represented by essays by some of their members.

The methods of work are somewhat varied. Dr. Clark Gapen, of Kankakee, issues sheets, each one of which is an synopsis of from twelve to twenty subordinate subject headings of a lecture, all details of which are to be given by the lecturer. At Rochester, Minn., the lectures in condensed form have been published this year as a book intended to cover a two years' course. This is to study by and for after reference. It avoids

note-taking, and provides for a recorded symmetry of the course, however often sickness, illness, or removal of lecturers may occur. Most schools, however, divide the course up among the members of the staff, who each, presumably, develops his department after his own ideas, the student taking notes usually, and thus getting various quantities of the matter presented. In many cases books are designated for reference and reading.

This is approximately the old medical college idea, which seemingly is being superseded by book study and recitation. In Westboro, Mass.; McLean Asylum, Mass.; Concord, N. H., and, perhaps, in one or two others, superintendents of training schools are appointed, thus attaining more of the form of general training schools. In some other of our schools, however, the one occupying the place designated as "head nurse" is not in very different authority.

In detail of work the general indication is of fair depth. Columbia, S. C.; Buffalo, N. Y.; McLean Hospital, Newark, N. J.; Essex County Hospital, Independence, Iowa; Rochester, Minn., and St. Paul, Minn., make a practice of sending nurses out to private cases of bodily disease. Probably others have also done this. Columbia, by Dr. Babcock, reports as many as forty-five cases so cared for in one year. Rochester, Minn., reported about twenty so cared for last year (obstetrical, surgical and typhoid nursing) together with the fact that five of their nurses are out as professionals.

The St. Lawrence Hospital, New York, exhibits the most elaborate system in its announcements, although not more extensive in its lectures than several others. The number of lectures given annually varies somewhat with different hospitals.

Dr. Cowles has questioned concerning the propriety of making a three-years' course instead of two years, and having uniform requirements by the hospitals, so that it shall be known exactly how much a diploma means. The establishing of some minimum of work and attainment will doubtless soon come, while the three-years' course may follow after, but needs a higher consequent pay to fully support it.

Of the class of students, it is, of course, true that hospitals must begin with the best of the material at hand. St. Lawrence, N. Y., specifies an examination for entrance. Doubtless this formality is of much value, though, perhaps, the common test of one or two months

trial, letting the applicant go if not suitable at the end of that time, can be made more efficacious, scholarship being but one of several qualifications needed. In either case, however, it is the standard which the superintendent lives up to, which tells, and he has a very important duty. The details of methods are not commonly published.

Making the school helpful to the interest of outside surgeons and practitioners is, in our experience, highly commendable, providing them with nurses from the school, either graduates or senior students.

Electricity, care of the operating room, preparations for surgical operations, urinalyses, practice in cooking special dishes for the sick are commonly taught on the infirmary flat, or the infirmary building of the hospital for each sex. Massage is taught usually as a special course in the senior year (if taught at all), in several cases, however being a part of the regular course. Obstetrical instruction can be made much more thorough by a half dozen extra lessons to the senior class. Several short post-graduate courses have been given here to volunteers, and there is always a volunteer interest in them, for they are all along made to feel that they have a profession, and that in a practical and not sentimental way.

Lady nurses alone seem to have graduated from two or three of the hospitals. Probably, however, a trial has never revealed any local difficulties in the way of including men. The lack of the idea of a permanent profession in the case of men is the only special drawback which we have found. The educational idea must be put forward, and the raising of wages to those who graduate and stay with the hospital, must help out as inducement. Doubtless, also, it will be a good rule to drop from the service those who do not pass the examinations.

It has been found in Rochester, Minn., that in about three-quarters of the classes, men and women can meet together with benefit to the nursing work. We have also encouraged the formation of a society among the graduates and seniors, the membership of which is entirely voluntary and the meetings conducted by themselves. Members are allowed to leave the society on a two weeks' notice, or come in by a vote of the members. They have at their meetings, music, readings, new reports, debates upon such topics as the advisability of

the "total prohibition of restraint," the "nursing of typhoid fever as compared with surgical work," etc., etc. The physicians have helped them out by giving talks upon bacteriology, microscopical work, etc. St. Peter State Hospital, Minnesota, originated this work in a society of a slightly different form and with good success. Evansville, Ind., mentions a lecture course for nurses, given by outside parties upon general subjects.

We consider, then, that we are to feel highly encouraged by this review; for it is not simply that one has to sit back and decide that he will have a training school, and it is done, but the pioneer in this work has to work against obstacles, has to work up the spirit of nursing, by being everywhere a part of it, by daily earnestness when students are themselves half-hearted, until the school has had its first graduates and successes to make the path easy.

A training school, then, is a kind of *prima facie* evidence of earnest work, and goes with and leads forward hospital methods. We do not consider that the hospitals for insane of this country can be truly charged on the whole as being behind those of Europe in their general clinical care of patients, even though it be claimed that we have not so many prominent pathological researches.

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**General Paralysis in Children from Hereditary Syphilis.** (*Gazette des hôpitaux*). M. Fournier in a report on a paper by M. Régis, relative to two cases of general juvenile paralysis from hereditary syphilis, comes to the conclusion that the diagnosis of general paralysis admitted of no doubts. Also that the hereditary syphilitic origin was equally certain. That these two facts went to support a relatively great number of analogous facts, made it certain that general juvenile paralysis is most always the result of hereditary syphilis (or sometimes acquired at a young age) just as the general paralysis of adults proceeds in the great majority of cases from acquired syphilis. In thirty seven cases of general infantile paralysis, syphilitic antecedents were certain in twenty-nine, and in eight they were probable. It is proper to add that in the two cases of M. Régis, the fathers of the patients were in a condition bordering on general paralysis, a condition which has been observed in several instances of general infantile paralysis. ONUF.

## Periscope.

### ANATOMICAL.

**The Theory of Neurons and Their Connection Simply by Contact.**—Renaut (*Med. Week*, March 8, 1895). The term neurons, which was first used by Waldeyer, has since been employed by Ramón y Cajal, and many other investigators, to designate a nerve cell in its totality, that is to say, with the inclusion of its axis-cylinder process and its protoplasmic branches; each neuron being an independent nerve element, a nervous unity. The axis-cylinder in its course sends out lateral branches, and ultimately terminates in more or less numerous free ends. The protoplasmic branches resemble the ramifications of a bushy tree, and also terminate in free filaments.

According to Ramón y Cajal, the neurons are connected not by continuity, so as to form anastomoses or reticula, but only by contact, that is to say, by genuine articulations.

In order to test these theories, Renaut studied the multipolar nerve cells and their reciprocal relations in the retina of rabbits and guinea-pigs, using Ehrlich's method of staining with methylene-blue. In preparations made in this manner, the nerve cells present ramifications of remarkable extent and complexity, whereas in the multipolar cells prepared by Golgi and Ramón y Cajal's method the ramifications are less complex and less bushy. In the former, indeed, the cells resemble a tree with all its branches, twigs and minutest branchlets, while in the latter the tree also exists, but apparently trimmed and shorn of its outer branches. It being evident, therefore, that the ultimate ramifications of nerve cells are not brought out by chromate of silver, he is inclined to think that Ramón y Cajal goes too far when he asserts that the protoplasmic processes invariably terminate by free ends.

By means of certain other permanent preparations made with methylene-blue, it is seen that some protoplasmic prolongations of a nerve cell provided with an axis-cylinder communicate with those of a cell possessing only protoplasmic prolongations, but no axis-cylinder, a variety of cells which have been found in the retina by Dogiel and Ramón y Cajal, and which the latter describes under the name of amacrine cells. There are, consequently, neurons which are connected and continued by some of their protoplasmic prolongations, though the connecting link is extremely slender, so slender, in fact, as to be imperceptible in a preparation treated by chromate of silver.

Lastly, in preparations fixed by picrate of ammonium in the presence of iodine, he has distinctly seen protoplasmic prolongations, springing from large ganglionic cells, run together so as to form a fine network on the inferior aspect of the basal plexus. Other peculiarities which appear to possess a certain importance have likewise been observed: in the first place, the undoubted existence of a hyaline capsule, enveloping the cell which is continued, but only for a short distance, along the protoplasmic prolongations.

The second important detail is the peculiar appearance, presented by the plexiform network formed by the protoplasmic prolongations. These appear to be beaded, owing to the fact that each one presents a series of

vacuolar swellings. In this beaded network the protoplasmic prolongations approach each other, and it is principally there that the articulation by contact is effected.

To sum up, it cannot be maintained that the neurons are never continuous, and, consequently, the theory of connection simply by contiguity does not appear to him to be quite exact.

On the other hand, it must be recognized that, if the articulation of the neurons with each other is mainly one of contiguity, it is accomplished in the special manner already alluded to. J. C.

**The Posterior Longitudinal Fascicle.**—(*Hinteres Langbündel, Faisceau Longitudinal Postérieur*). Van Gehuchten (*Bulletin de l'Académie Royale de Médecine de Belgique*, 1895, No. 2).

The posterior longitudinal fascicle is a bundle of nerve fibres which passes from the proximal extremity of the mid-brain through the whole extent of the peduncle (Stamm) to the middle parts of the medulla oblongata, where it becomes mixed up with the fibres of the reticular formation. The said bundle is situated at both sides of the "raphe" ventrad from the aqueductus Sylvii and from the median part of the floor of the fourth ventricle. Van Gehuchten has studied the course, origin and connections of this fascicle in the brains of trouts by use of the methods of metal impregnation (Golgi's and Ramon y Cajal's methods). The result of his researches leads him to the following conclusions:

1. The posterior longitudinal fascicle is formed exclusively of descending fibres. Consequently it must be considered as a motor nerve bundle.

2. It extends from the proximal part of the mid-brain to the anterior column of the spinal cord. It increases in volume in caudad direction, by taking up new fibres from the adjoining gray masses.

3. Its most proximal fibres take their origin from cells which form a group situated at both sides of the median line, ventrad from the ependymary epithelium and dorsad from Meynert's fascicle; for a certain distance this gray mass which Van G. calls the superior nucleus of the "posterior longitudinal fascicle" accompanies the nucleus of the third nerve, being situated dorsad to it.

4. The remaining fibres composing the posterior longitudinal fascicle originate from cells scattered in the "trunk" (Stamm), most of which are situated along the descending root of the fifth nerve and at the level of the nucleus of the seventh nerve.

5. In their course through the "trunk" the fibres of the post-longitudinal fascicle give off numerous collaterals which ramify in the adjoining gray masses, especially in the nuclei of the third, fourth and seventh nerves and in the anterior horns of the spinal cord, thus connecting themselves with the cells which give origin to peripheric motor fibres.

6. Most of the fibres of the posterior longitudinal fascicle are direct (not crossed) fibres, part of them, however, originate from cells situated on the opposite side of the median line—crossed fibres.

7. Of the collaterals, which the fibres of the posterior longitudinal fascicle give off in their course, the larger part remain on the same side (direct collaterals), part pass the median line to ramify in gray masses of the opposite side (crossed collaterals).

8. The posterior commissure has no connection with the posterior longitudinal fascicle; that is, no fibres or collaterals of the latter are seen becoming fibres of the posterior commissure. ONUF.

**Reil's Fascicle and the Cerebral Cortex.**—By Prof. Déjerine and Mme. Déjerine (*Gazzetta degli Ospedali e delle Cliniche*, 1895, No. 46).

Based upon the result of the examination of nineteen brains affected with cortical lesions, the authors conclude that Reil's median fascicle (fillet) consists for the most part of fibres, the cells of origin of which are situated in the nuclei of Goll's and Burdach's (cuneate) columns. They

do not believe it proved, that the thalamus contains cells giving direct origin to part of said fibres. They find, however, that contrary to Flechsig's and Hoessel's views, the fillet does not present a direct and uninterrupted connection between the cortex on one side and Goll's and Burdach's columns on the other, but that this sensory bulbo-cortical tract involves two neurons, a bulbo-thalamic, represented by Reil's median fascicle (fillet), and a second one connecting the thalamus with the cortex.

ONUF.

## PHYSIOLOGICAL.

**The Physiological Action of Massage.**—In the February 1st number of *Lo Sperimentale* (Florence) Dr. Carlo Colombo describes the action of massage upon the various secretory organs of the body and arrives at the following conclusions:

a. Influence of massage upon the secretion of gastric juice.

1. Without massage he was only able to collect in two hours time about 15 ccm. of gastric juice, which was collected in a small bladder. After massage he was able to collect in two hours' time forty cubic centimetres of juice, of which one part was mucus; the remainder was pure gastric juice.

2. A massage of five minutes made but little difference in the secretion of the juice, but if continued for fifteen minutes the maximum secretory intensity is reached, and the proportion of hydrochloric acid and pepsin is not increased.

b. Influence of massage upon the secretion of bile.

1. The quantity and quality of the bile is not changed perceptibly after ten minutes of friction over the hepatic region, and of rubbing over the inferior border of the liver.

2. After ten minutes of concussion the quantity of bile is increased considerably in four hours. The biliary salts of soda and cholesterine are more abundant.

3. Twenty-five minutes of friction produces the same results as ten minutes of concussion.

4. The best effect of the massage would be to combine ten minutes of concussion, with ten of friction.

c. Action of massage upon the secretion of saliva.

1. The submaxillary glands are most sensitive to massage; after five minutes of manipulation the secretion is increased.

2. After ten minutes of massage the greatest intensity of flow is reached, in the submaxillaries as well as the parotids.

3. The saliva secreted, the result of the massage is similar to that which is obtained when the chorda tympani is excited.

d. Influence of massage upon the secretion of urine.

1. The quantity of urine was considerably increased after a local application of massage over the kidneys.

2. The specific gravity was not diminished, but there was an abundant supply of renal epithelium in the sediment and a trace of albumin was observed.

e. Action of massage upon the secretion of spermatazoa.

1. The action of massage upon one of the testicles produced double the quantity of testicular secretion than from the one not massaged.

KRAUSS.

**On the Time of Appearance of Secondary Degeneration in the Single Tracts of the Cord.**—Dr. K. Schaffer (*Neurolog. Centralbl.*), 1895, No. 9.

Schaffer performed total transverse section of the cord on cats. The animals were killed on the 3d, 4th, 5th, etc., day after the operation respectively. The cord was examined after Marchi's and Algeri's method.

1. The first degenerative changes were observed on four days after the experiment. In ascending direction there was beginning degen-

eration of Goll's columns. In descending direction there was degeneration in Lowenthal's descending marginal fascicle of the anterior column and in the intermediary fascicle of the lateral column (*faisceau intermédiaire du cordon latéral* of Lowenthal).

2. On the sixth day beginning degeneration of the lateral cerebellar tracts (*Kleinhirnsseitenstrangbahn*) was present in addition to the degenerations mentioned under 1.

3. On the 14th day degeneration of the lateral pyramidal tracts is noticed. The highest intensity of florid decay of the medullary sheaths is reached on the 4th day in Lowenthal's fascicles, on the 12th to 14th days in Goll's columns and the cerebellar tracts.

4. The descending degeneration of the posterior columns at the locality of Schultze's comma is to be seen on the 4th day in slight extent; it does not increase later. The temporal succession of the degeneration in the various tracts accordingly corresponds to that of the development of medullary sheaths. Those tracts of the fibres of which receive their medullary sheaths first degenerate first. From Flechsig's researches it is known that the fibres of Goll's columns are supplied with medullary sheaths at the end of the sixth month, those of the cerebellar tracts (*Kleinhirnsseitenstrangbahn*) towards the beginning of the seventh and the pyramidal tracts towards the end of the ninth month, or in other words the same temporal succession as they degenerate. ONUF.

**Acute Mania.**—DeWitt (*Lancet-Clinic*, June 22, 1895). The medical treatment of these cases is very simple, and can be disposed of in few words. To procure sleep and quiet is perhaps the greatest desideratum, and I know of nothing so certain in its action as chloral hydrate, given in 40 or 60 grains. It may be given alone or combined with one of the bromides. Bromidia I have always found very reliable. It is almost certain to quiet and produce sleep. Occasionally one meets with cases that resist the influence of chloral even in large repeated doses; here opium or some of its derivatives either given alone or in connection with the chloral, will be found of service. If hypodermically administered, not less than  $\frac{1}{2}$  gr. should be given. Small doses only excite the patient, and do more harm than good. Hydrobromate of hyoscine has some advocates. The milder hypnotics, such as sulfonal, chloralamid, etc., are not to be thought of in these cases; they are practically inert, and do no good. J. C.

#### PATHOLOGICAL.

**The Pathological Anatomy of Chronic Chorea.**—*Gazzella degli Ospedali e Delle Cliniche*, Mar. 15, 1895. At a meeting of the R. Academy of Medicine of Rome, February 25, 1895, Dr. Bignami reported the results of his pathological studies on two cases of chronic chorea in adults, without hereditary history. In one case he found sclerosis of the cerebral arteries, slight thickening of the pia, and atrophy of the gyri in the frontal and parietal lobes, which presented microscopically multiple foci of disseminated chronic encephalitis.

In the other case he found no alterations of the meninges, atrophy of the convolutions, numerous miliary patches, sub-cortical of sclerosis especially in the frontal lobes, a large focus of sclerosis in the external nucleus of the thalamus which diffused itself into the sub-thalamic region interrupting somewhat the radiating fibres of the internal capsule; much smaller foci were also found bilaterally and symmetrically in the red nucleus. Microscopically these foci are composed of a fine fibrillary tissue not in nuclei; cellular elements are also present having large rounded or oval nuclei, and abundance of protoplasm. KRAUSS.

**Vertigo.**—One of the most valuable contributions to our knowledge of the puzzling symptom known as vertigo has recently come from the pen of Mendel of Berlin. He says that various theories have



been formulated by medical men on the subject of vertigo, and it would be well, to come to an understanding as to what this condition really is. In the first place, vertigo is almost invariably ushered in by ocular phenomena. In some cases the patient has the sensation of seeing the surrounding objects whirling around himself, and experiences a tendency to fall either in the same direction as the objects seemingly turn, or toward the other side; at other times, it seems to the patient as if the objects about him were approaching or receding, rising or falling.

This instability of the external world is immediately followed by a sensation of oppression and sinking, resulting in static disturbances, and the patient begins to stagger and collapse. Then follow various secondary phenomena, such as pain in the back of the head, noises in the ears, ephemeral deafness, vomiting, slowness of the pulse, diaphoresis, etc.

There are four degrees of vertigo; in the first and lightest form, only ocular symptoms occur, accompanied by *angor* which rapidly disappears; in the second, there is a sensation of disturbed equilibrium; in the third, the equilibrium is really upset and the patient falls; in the fourth, these symptoms are accompanied by the above-mentioned secondary phenomena.

The pathognomonic clinical sign of vertigo is disturbance of the equilibrium. Maintenance of the equilibrium is intimately connected with the sense of touch, kinesthetic sensations, and sight. Whether hearing has anything to do with it, is still a subject of controversy.

All static disturbances are not symptomatic of vertigo. Abolition of the sensorium results in a loss of the equilibrium, which has nothing in common with vertigo; the fear experienced on being placed at a considerable height is not vertigo properly so called, but rather a psychological process which only secondarily affects the organs of equilibrium; the same is true of agoraphobia.

True, vertigo is always associated with a disturbance in the musculature of the eyes. Vertigo is the first symptom complained of by patients who suffer from paralysis of the ocular muscles, whether this paralysis be peripheral or nuclear. On several occasions I have even seen vertigo precede the paralysis.

On investigation of the causes of ocular vertigo, with special reference to the supply of blood to the muscles of the eye, I have found that the nuclei which preside over the movements of the ocular muscles receive their blood from the posterior cerebral artery through fine terminal branches, not anastomosing with each other. The result of this arrangement is that the slightest disturbance in the central circulation immediately detracts from the supply of blood to the nuclei of the ocular muscles.

Every pathological process which develops a focus of disease in the brain will necessarily influence the cerebral circulation and, in consequence thereof, be associated with vertigo, especially when this focus occupies a posterior cerebral fossa.

Lastly, vertigo is the earliest and most prominent sign of a morbid change in the blood vessels of the brain, although arteriosclerosis is not accompanied by vertigo unless its evolution is very rapid, a process of adaptation, taking place otherwise.

The treatment of vertigo must be adapted to its cause. In cerebral arteriosclerosis iodide of potassium in small doses, continued for months, and, in addition, camphor and quinine, to re-enforce the action of the heart, seems to give the best results. With the same object in view, alcoholic beverages in moderate quantities should be prescribed to patients who suffer from this affection. In recapitulation he says, vertigo consists in a group of symptoms, determined by a disturbance in the sense of equilibrium, due to defective function of the ocular muscles. This defect itself may be of peripheral or central origin. J. C.

## CLINICAL.

**Fever of Nervous Origin.**—Duvernet (*Gazette des Hôpitaux*) publishes the case of a woman, aged 58, who had previously been healthy and never exhibited symptoms of hysteria. On the 12th of May, 1891, she was taken sick with a moderate fever, malaria, headache and insomnia. These symptoms lasted one month, when a mild diarrhoea appeared. On June 15th the temperature suddenly rose, accompanied with great agitation. The morning pulse ranged from 80 to 90 and rose at night to 120. The patient also had a chill followed first by a dry hot skin and then a profuse perspiration. The axillary temperature was taken several times daily, with every precaution to avoid fraud. On the evening of the 21st it was 40°6 C. The following morning 37.7 C. For some days following the evening temperature did not go above 39.5 C. and in the morning it remained at 37.7 C. This continued till July 6th. On the 7th the evening temperature suddenly rose to 41 C. Then for a few days it continued at 38.5 C in the morning and 40 to 41 C. or more in the evening. There also developed subsultus tendinum, tympanites, and some lenticular rose-colored spots which made the author think of typhoid fever, but later he concluded that these symptoms were of nervous origin. During the following four months the morning fever continued at 38.5 C. and the evening at 41.1 C.; exceptionally it would mount to 42.7, 43.4, and 43.7, C. The patient made a sea voyage and then later went to Rome and spent the winter, but all this time the fever continued. Finally in May, 1892, it disappeared, but the diarrhoea, insomnia, and tendon twitchings have continued without interruption during the years 1893 and 1894. The treatment has been unsuccessful. Quinine, all the antipyretics and bromides have failed. Opium and chloral have appeared more useful. Sulfonal alone has seemed of any value for the sleeplessness. Cold lotions and warm baths were without effect.

Duvernet considers this a rare case of nervous fever in a now hysterical patient. It shows, he says, that the danger of hyperpyrexia depends less upon its intensity than upon the cause which produces it.

FREEMAN

**The Prognosis of Acute, Non-purulent Encephalitis.**—Oppenheim (*Deutsche Zeitschr. f. Nervenheilk.*, Vol. vi., Part 5 and 6, 1895).

The conception of encephalitis from a symptomatic point of view is an incomplete one and very difficult to define, but with one form of this disease we are sufficiently familiar, clinically and anatomically to separate it and differentiate it from other types of the disease. And the term to which Oppenheim refers by this opening sentence is the acute non-purulent encephalitis, a form which almost always ends in complete or partial recovery. It differs materially in its etiology, its course and prognosis from other forms of encephalitis, and is characterized in a way by its sudden onset, its acute course (although the author reports a case in which the disease ran a subacute course and ended in recovery) and its favorable termination.

The history of the disease, as it occurred in five patients, is given. The first, a girl of sixteen, of good family and personal history, without history of recent acute disease, was taken with severe headache immediately after having made a short railway journey; accompanying the headache was loss of appetite, thirst and intense feeling of heat in the head. About a week later she noticed that it was impossible to speak. In this patient there was no vertigo, spasms or paresis of the extremities. Although consciousness was never entirely lost, there were times when it was not fully intact. When admitted to the hospital the bodily temperature was slightly elevated; the patient answered questions partly, incorrectly, or not at all; percussion of the head seemed to annoy

her; pupils were equal and responded quickly to light; no stiffness of the neck; although it was apparently distressing for the patient to bend the head forward so that the chin struck the chest. No paralysis, and knee-jerks well preserved. A week after her entrance in the hospital, and a month after the first appearance of the symptoms, ophthalmoscopic examination showed left optic neuritis in an incipient stage; right eye normal. At this time no headache and no pain on movement of the head. Considerable stupidity, some things are understood, others not. Uses words wrongly, instead of chin, says nose; instead of mouth, says ear, etc. No disturbance of sensibility or motility; pulse rather frequent; sphincters normal; three months later the patient had recovered.

The writer gives in detail the history of three other patients, and they have in common the localization of the process in the brain. Always in the foreground was the aphasia. This did not develop as it does after an apoplectic insult or an encephalomalacie, but after a headache lasting ten to twelve days, and after the beginning of an illness whose onset was characterized by malaise, fever, stupidity and the then aphasia. The absence of motorial irritation and appearance of paralysis, the deep disturbance of consciousness and the spinal symptoms, as well as the early appearance of aphasia spoke against the diagnosis of meningitis.

In the first and second cases absolutely no etiological factor could be attributed as the cause of the disease; in the second and third cases it would seem that influenza could be. In none of the cases was there any evidence of syphilis.

The disease must be distinguished from disseminated sclerosis, which the author thinks runs not infrequently an acute course and ends in recovery. He believes that multiple sclerosis is probably in many cases nothing more than a combination of myelitis and encephalitis. In acute non-suppurative encephalitis, very abrupt onset, severe symptoms, including high temperature, are signs of danger, while a slow onset, low temperature and protracted course are of good omen. J. C.

**Influenzal Meningitis.**—Cornil (*Semaine Médicale*, May 10, 1895), describes a patient who was admitted into his wards for influenza, complicated with cerebral symptoms.

The patient was a woman, forty years of age, non-alcoholic, who, on April 10 last, was seized with headache and general lassitude, followed in a few days by fever, prostration and drowsiness, which soon passed into coma, accompanied by stertor. On admission she presented hemiplegia of the limbs and upper area of the facial on the right side, incontinence of urine and faeces, etc.

She died on April 24. At the autopsy, the pia mater was hard and infiltrated with an abundant, yellow, opaque liquid. In the right half of the brain there was a small hæmorrhagic focus, situated in the grey matter, and another in the first occipal convolution of the same side, which also was subcortical.

This was, consequently, a case of encephalo-meningitis, predominating in the convex part of the brain. Bacteriological examination did not reveal the presence of the influenza bacillus, though no conclusion can be drawn from its absence, as it is quite doubtful whether it ever passes into the blood. J. C.

**A Constant Sign in Incipient Meningitis.**—Simon (*La France Méd.*, March 29, 1895), in his *clinique*, called attention recently to the fact, that tubercular meningitis always presented no symptoms during the onset, the importance of which he considered indisputable. The sign is a disharmony, viz., an irregularity (dissociation) of the respiratory movements of the diaphragm and the thorax, which sets in during the first days of meningitis, and which can be of great assistance in detecting it, even in the most typical and insidious cases.

MACALESTER.

## THERAPEUTICAL.

**Sulfonal in Insanity.**—(*Die Therapie der Gegenwart*, June 1895). In the Marburg clinic for mental diseases Schedtler administered this drug to forty-one insane females. As a rule the usual doses were given. Restless and apprehensive cases received from .05 to 0.3 gms. several times in the 24 hours. Disagreeable effects were repeatedly observed. The objective symptoms which in the insane he considers especially important are in mild forms of sulfonal intoxication—drowsiness, pallor and nausea shown by frequent spitting and uneasiness, also vomiting, staggering gait and sometimes diarrhœa. If the sulfonal be discontinued when these characteristic symptoms first appear, the patients without exception recover completely. Permanent ill-effects were never observed. The dose necessary to cause intoxication fluctuated between wide limits, and appears to depend upon individual idiosyncrasy. The great majority of the patients to whom this remedy was regularly given for a long time bore 1.0 gms. to 2.0 gms. a day without any unfavorable influence. In two cases, however marked symptoms were observed. The first received a relatively large amount of sulphonal in a short time (from June 8th to July 5th 68. gms.), and suffered from vomiting and diarrhœa. The patient presented a miserable, dejected appearance. Some sugar was found in the urine but no albumin. Upon discontinuing the sulphonal the condition improved and recovery took place. The symptoms in the second place pointed to cerebral toxæmia. There was also a cutaneous eruption present. Even when this drug is well born, S. advises that it should never be given in daily doses of from 2 to 3 gms. more than several months, without discontinuing it from time to time, or changing it for another narcotic. By observing carefully these rules, cases of severe intoxication have not occurred in the clinic for the past two years. FREEMAN.

**Trional Poisoning.** (*Die Therapie der Gegenwart*, June, 1895.) H. Reinicke reports a case of chronic trional poisoning, which developed in spite of the most careful administration of this remedy. From time to time it was discontinued, the diet was guarded, and the action of the kidneys and bowels closely watched. In all, the patient received 40 gms. in 107 days. Suddenly she complained of headache and dizziness. Two days later, although the trional was stopped, nausea and diarrhœa with thin watery blood stained passages came on. Four days afterwards the urine voided was of a dark red color, and contained albumin and numerous hyaline and granular casts. Two previously reported cases also in women, had a lethal ending. The profuse bloody diarrhœa in the beginning of R.'s case is of interest, as in the cases previously reported constipation was observed. It is apparent that the bloody diarrhœa, which disappeared immediately upon discontinuing the remedy, was caused by the trional exerting the same injurious influence on the gut as on the kidneys. The interest of this case lies in the fact that only a relatively small amount of trional was administered, and yet under the most careful management poisoning occurred.

FREEMAN

THE  
**Journal**  
OF  
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AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-first Annual Meeting, held in Boston, June 5, 6  
and 7, 1895.*

*(Continued.)*

*Prize Essay, for which the American Neurological Association  
Awarded \$200.00 at its Annual Meeting,  
June 5, 6 and 7.*

THE BIOLOGICAL AND MORPHOLOGICAL CON-  
STITUTION OF GANGLIONIC CELLS, AS  
INFLUENCED BY SECTION OF THE SPINAL  
NERVE ROOTS OR SPINAL NERVES. TO  
WHICH IS APPENDED SOME REMARKS ON  
LOCALIZATION.

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ALTHOUGH observations on ascending changes fol-  
lowing lesion of peripheric nerves or of their  
roots are becoming more and more numerous, a  
perusal of the literature of this subject shows that a  
unanimity of opinion as to the extent and nature of these  
changes has not been reached. Indeed, one of the latest  
investigators, Vanlair, has attained results which seem  
to contradict all observations made heretofore. He  
states that unilateral section of a spinal nerve, whether  
a regeneration of the latter took place or not, is in no  
case followed by any alterations in the structure of the

spinal cord. His conclusions are based upon the results of numerous neurotomies and amputations performed on full grown dogs which were killed from six months to two and a half years after the date of operation.

I mention these facts in order to demonstrate that the final word has not yet been spoken and that, therefore, I may be excused for taking the subject up again.

The older literature on this subject has been exhaustively treated by Homen, which allows me to make my review of it rather brief.

The anatomical material on which previous authors based their conclusions was obtained in various ways. The largest number of publications are descriptions of the alterations found in the spinal cord and nerve stumps of persons who had previously suffered the loss of a limb by amputation.

In one case (Edinger) the amputation had taken place before the birth of the patient, that is, the child was born without a forearm. In another case (Strümpell) compression of the cauda equina in a man furnished the pathological material. Other researches have been made by aid of the experimental method, comprising neurotomies, neurectomies, ligation of nerves, amputations, sections of nerve roots and similar operations.

As to the alterations following these various injuries, I shall first mention those found in the gray substance of the spinal cord, and in order to simplify the matter, I shall give some tables of the observations:

I. The gray substance was found entirely normal.

AUTHOR.	MODE OF OPERATION.	RESULT.
Buffalini et Rossi. Vanlair.	Section of spinal roots. Amputations and neurotomies.	No alterations when the lesion was unilateral.

II. Both the anterior and posterior horn of the operated side showed a lessening of volume and change of shape.

AUTHOR.	MODE OF OPERATION OR PATHOLOGICAL CAUSE.	
Hayem. Hayem et Gilbert. Vulpian.	Tearing off of sciatic nerve. Amputation. Amputations in man and neurotomies of sciatic nerve of rabbits.	} Chiefly anterior horn affected.
Reynolds.	Amputation	
Edinger.	Amputation in utero.	
Kahler and Pick.	Amputation	

Mayser.	Section and tearing off of sciatic nerve.	Chiefly posterior horn affected.
Marinesco.	Amputation.	Results different in different cases.
Dejerine et Mayor.	Amputation.	Posterior and anterior horn affected in equal proportion.

III. The alterations were limited to the anterior horn, while the posterior horn was found perfectly normal.

AUTHOR.	MODE OF OPERATION OR PATHOLOGICAL CAUSE.
Dreschfeld.	Amputation.
Dudley.	Amputation.
Genzmer.	Amputation.

IV. The changes were exclusively or nearly exclusively confined to the posterior horn and sensory tracts, and the anterior roots were normal or nearly so.

AUTHOR.	MODE OF OPERATION OR PATHOLOGICAL CONDITION.
Max Joseph.	Section of spinal nerves and of roots of spinal nerves.
Friedländer and Krause.	Amputations.
Bignamini and Guarnieri.	Amputation of thigh.
Kahler.	Section of spinal roots.
Strümpell.	Lesion of cauda equina.
Homen.	Experimental amputations.

The authors mentioned in the last table firmly maintain that section of motor fibres in the anterior roots or in the peripheric course of spinal nerves does not cause any ascending alterations of these fibres or of the nerve cells from which they take their origin.

Friedländer and Krause conclude that if the motor paths became atrophic in ascending direction, the atrophy should include the anterior roots, which, they state, was never the case in their observations. They, therefore, arrive at the conclusion that the ascending atrophy following the amputation of a limb is exclusively confined to the sensory tracts.

Max Joseph, Strümpell and Bignamini and Guarnieri confirm F.'s and K.'s opinion. Homen inclines very much to the same view, although he expresses himself somewhat less positively.

Hayem's conclusions are: "L' atrophie cicatricielle

produite dans le faisceau postérieur de la moelle en dehors du cordon de Goll par l'arrachement d'un nerf détermine une atrophie très rapide des cellules nerveuses qui porte surtout sur le groupe du tractus intermedio-lateralis (postero-lateral group)."

This conclusion is rather peculiar since Hayem distinctly states that the anterior roots were also atrophic and that throughout the whole area of gray substance the nerve cells were atrophied. It would certainly seem more natural to connect etiologically the atrophy of the nerve cells in the anterior horn with that of the anterior roots instead of with that of the posterior roots. Homen, Friedländer and Krause, who examined the spinal cord of persons or dogs on whom amputation had been performed, also found atrophy or lessening of the number of cells in the postero-lateral group, but found only quite insignificant alterations (if any) in the cells of the other groups of the anterior horn. The atrophy of the postero lateral group had to be explained, of course, considering that the anterior roots were found normal. The explanation given by them is, that the cells of this group are most probably sensory, a view which finds some support in Lenhossek's observation. The latter's conclusions were based on observations made by the aid of Ramon y Cajal's method. He saw fibres originate from cells in the lateral part of the anterior horn and pass through the spinal cord, posterior root and spinal ganglion without becoming connected with cells of the latter.

Marinesco and Reynolds oppose the theory that the postero-lateral group has sensory functions.

That alterations of cells in the postero-lateral group have, indeed, been much more frequently and more constantly observed than changes of the cells of any other group, will be shown by the following table:

Atrophy or disappearance of nerve cells of the anterior horn was found by:

AUTHOR.	MODE OF OPERATION OR PATHOLOGICAL CAUSE.	CELL GROUPS AFFECTED.	
Dudley.	Amputation.	The antero-lateral and postero-lateral groups are those in which the difference is most evident.	Chiefly antero-lateral and postero lateral groups.



Friedländer & Krause.	Amputation.	The postero-lateral group is exclusively affected.	Exclusively or nearly exclusively postero-lateral groups.	Predominantly postero-lateral group.
Homen.	Amputation.	The postero lateral group is nearly exclusively affected.		
Vulpian.	Amputation & section of sciatic nerve.	The postero-lateral group is predominantly affected.		
Hayem.	Tearing off of sciatic nerve.	Rapid atrophy of nerve cells, especially of the postero-lateral group.		
Mayser.	Section & tearing off of sciatic nerve.	The postero-lateral group is entirely absent.	Chiefly antero-internal group.	
Marinesco.	Amputation of thigh or arm.	The postero-lateral group is principally affected.		
Hayem and Gilbert.	Amputation of thigh or arm.	The gray substance is nearly entirely deprived of nerve cells.		
Reynolds.	Amputation of thigh or arm.	All groups affected.		
Dejerine and Mayor.	Amputation of thigh or arm.	Only in one of four cases was there a lessening of the number of cells. It was stated in all three groups, but chiefly in the antero-internal one.	Chiefly central group.	All groups.
Kahler.	Amputation of thigh or arm.	Cell changes in all groups, but chiefly is the central one.		
Vaulair,	Section of sciatic nerve.	Central group affected in one case.		
Erlitzky.	Amputation.	Lessening of the number of cells of the anterior horn.		
Genzmer.	Amputation.	The number of nerve cells of the anterior horn is lessened.	Chiefly antero-internal group.	
Dickson.	Amputation.	The number of nerve cells of the anterior horn is lessened.		
Edinger.	Amputation during fetal period.	Destruction of cells of the anterior horn.		

It is noteworthy that all cases in which the postero-lateral group was affected were taken from the spinal cord of persons or animals on whom amputations or section of peripheric nerves had been performed. In none of them had isolated section of the posterior or anterior roots been made. Singer and Muenzer and Rossolymo cut the posterior roots separately without injuring the

anterior roots, but none of these authors mentions alterations of the cells of the postero-lateral group as a consequence of this operation. Rossolymo states that there was a decrease of the number of cells in the posterior horn of the operated side, but he says nothing about the postero-lateral group.

There is consequently sufficient reason to make it questionable whether the postero-lateral group has sensory functions. It must also be kept in mind, that in many cases of amputations or section of the sciatic nerve the principal alterations were found in the central (Vanlair) or antero-internal (Dejerine and Mayor), or antero-lateral group (Dudley, in whose case, however, the postero-lateral group was also much atrophied). Accepting Friedlander and Krause's theory, these groups must, therefore, have sensory functions, a conclusion which contradicts too seriously the findings in progressive muscular atrophy, poliomyelitis anterior, etc., to be accepted.

The condition of the affected nerve cells has been described as simple atrophy by many authors, as a degenerative atrophy by others. Besides the complete disappearance of cells which, of course, signifies the highest degree of atrophy or degeneration, decrease in size of the cells, shrinking or disappearance of the processes, alterations of the nucleus and abnormal staining have been observed. I have met with only three cases in the literature where hypertrophic or pseudohypertrophic conditions of presumably motor cells were mentioned to be the secondary effect of a lesion. Of these three cases only one can properly be used for comparison, as in the two others the lesion which called forth said condition was situated in the spinal cord itself. However, all three cases are interesting enough to be mentioned:

The first case is described by Mayser and relates to the spinal cord of a rabbit, in which immediately after birth a segment of the spinal cord in the dorsal region had been extirpated. Just how long after the operation the animal was allowed to live is not specified. The lumbar enlargement of the cord showed the following characteristics: "The gray substance has remained in an undeveloped state, the nerve cells are all larger than normal cells, less stained, pale; with soft, so to say, hydropsically swollen protoplasm and enormously swollen nuclei."

The second case, reported by Kahler and Pick, was a patient whose cervical spine had been fractured. The spinal cord was completely cut through at the height of the injury. The patient died twelve weeks after the accident.

In the lumbar enlargement the alteration of the white substance is confined to the changes caused by simple degeneration of the pyramidal tracts. Otherwise the white and also the gray substance appear normal with the exception, that some of the cells in the anterior horn have a pale, swollen appearance.

The third case is also reported by Kahler and Pick. The primary lesion was amputation of the left leg in the lower third of the thigh. Death was caused by cyanide of potash poisoning eighteen years after the date of injury. In the lumbar enlargement the following remarkable condition was present:

"There is a peculiar type of nerve cells in the anterior horn of the left side. These cells are decidedly enlarged by an increase of their contents. This is demonstrated by their round shape, and especially by the fact that the parts which are situated between the processes of the cells are not concave as in normal cells, but convex."

The cells mentioned appear paler and stained much more evenly by carmine than the normal cells of the other side. They have a homogeneous colloid appearance. The nuclei with very distinct nucleoli are located quite peripherically. They show off very distinctly, and in some sections appear like a projection attached to the cell. The mass of pigment is crowded towards the base of a process. The processes themselves show a normal appearance.

Nowhere are there signs of an inflammatory process to be seen.

The largest number of cells of the described type are found in the external (postero-lateral) group; they are less numerous in the medial (central) group, while there are none in the anterior group.

Aside from this hypertrophic or pseudohypertrophic condition one notes atrophy and disappearance of cells, that is, a lessening of the number of cells in all groups of the anterior horn. Atrophy and hypertrophy of cells alternate in such a manner, that evidently the same cause (amputation, section of the motor nerve fibres) produced hypertrophic (?) changes in part of the "motor"

cells, atrophic in the others, unless the hypertrophy represents the primary stage of the changes which in other cells led to atrophy or complete disappearance.

The cases related heretofore—with the exception of the two cases of injury to the spinal cord—concern lesions of spinal nerves or of their roots. Lesions of cerebral motor nerves have been produced in different manners, viz., by section, excision, ligation, tearing off from the base of the brain, etc. (Gudden, Forel, Mayser, Ganser, Darkschewitsch and others). They always effected secondary alterations in the nerve cells from which the injured motor fibres took their origin. These alterations consisted in a shrinkage, which in the newborn led to a complete disappearance of the cells, while in the adult it was less pronounced; the intensity of the retrogressive process varying with the distance of the point of lesion from the motor centre in question, with the age of the animal and with the length of the interval between the injury and the death of the animal. Cell changes of a hypertrophic character in the centres of cerebral motor nerves have not been reported. Of the structural changes which can be discerned by the use of Nissl's method I shall speak later on.

So much for the observations on the structure of nerve cells as influenced by lesions of motor nerve fibres. It is not my intention to speak now of secondary degeneration occurring in the peripheric portion of severed motor fibres, but I wish to report the changes that have been noticed in the central (ascending) portion of the latter.

The following authors found the motor fibres of the stump, or at least the anterior roots corresponding to the injured nerve entirely normal:

AUTHOR.	MODE OF OPERATION OR PATHOLOGICAL CAUSE.
Vulpian.	Amputation and section of sciatic nerve.
Friedländer and Krause.	Amputation.
Kahler.	Section of both the anterior and posterior roots.
Dejerine and Mayor.	Amputation.
Max Joseph.	Section of nerves and of roots.
Bignamini and Guarnieri.	Amputation.
Strümpell.	Lesion of the cauda equina.

Dejerine and Mayor found changes in the central nerve stump of amputated persons, but stated that these

changes did not reach up to the spinal cord, the roots being normal. Vanlair concludes that after an amputation or a neurotomy (whether regeneration takes place or not), an increase of the fine nerve fibres (fibres grêles) occurs which must not be ascribed to an atrophic or degenerative process, but to a proliferation of the pre-existing fibres. This process of proliferation was found to diminish in ascending direction and seemed to reach its upper limit at the point of emergency of the nerve from the spinal cord.

Other authors have found distinct atrophic or degenerative changes of the anterior roots of spinal nerves or of the intramedullary roots of cerebral nerves, as will be seen from the following table :

AUTHOR.	MODE OF OPERATION OR PATHOLOGICAL CAUSE.	CHARACTER OF CHANGES	
Hayem.	Tearing off of sciatic nerve.	Atrophy.	
Bérard.	Amputation.	Attenuation of the roots. No microscopical examination made.	
Hayem et Gilbert.	Amputation.	Atrophy of extra medullary anterior and posterior roots.	
Dickinson.	Amputation.	Wasting of the nerve roots, especially of posterior.	Atrophic changes.
Dudley.	Amputation.	Fibres of anterior roots less numerous.	
Reynolds.	Amputation.	Anterior nerve roots smaller.	
Edinger.	Amputation in ultero.	Atrophy of sensory and motor roots.	
Genzmer.	Amputation.	Anterior roots thinner and containing less fibres	
Marinesco.	Amputation.	Distinct atrophy of the anterior roots in one case.	
Forel.	Lesions of cerebral motor nerves.	Marastic shrinkage of fibres of varying intensity, according to age of animals, etc.	
Marinesco.	Section of pneumogastric nerve in rabbits.	Decay of the medullary sheaths demonstrated with Marchi's method by the impregnation with numerous black granula.	Degenerative changes.
Darkschewitsch.	Lesions of spinal and cerebral motor nerves.		

In opposition to the view of Homen, Cossy and Dejerine and others, that if ascending changes took place as a consequence of injuries to motor nerve fibres they were of a purely atrophic nature, Forel had come to the following conclusions:

1. The motor nerve degenerates in the adult in both directions, the motor cells of its physiological centre being involved in the process of degeneration. This takes place when the nerve is severed at the base of the brain.

2. Section of the motor nerve in its peripheric course, provided that a regrowth of the nerve fibres into the peripheric stump is prevented, causes a slow marastic shrinkage of the nerve fibres in the central stump, and also of the nerve cells from which they take their origin.

3. There is no qualitative, but only a quantitative difference between secondary degeneration and the atrophy affected after Gudden's method. Both are actually processes of the same nature and consist in a necrosis of the injured element (neuron) in its whole extent.

Since Marchi's method became known Singer and Muenzer, Darkschewitsch, Marinesco and Moxaew (Mojieff) have applied it to verify, which view was correct; that is, whether the process occurring in the central stump of severed peripheric nerves should be called atrophy or secondary degeneration in the sense of Waller. As it is, the method used that decides Singer and Muenzer to oppose Forel's conclusions, I may be excused for giving a brief description of Marchi's method in its improved form.

The fresh specimen is cut into pieces not exceeding one cubic centimetre in size. These pieces are hardened in Müller's fluid for at least one week, but in no case longer than three months. Subsequently they are transferred to a mixture of one per cent. osmic acid (one part) and Müller's fluid (two parts) for from five to eight days, then washed out thoroughly in water during several hours and treated in the manner required for celloidin drenching.

By this treatment those nerve fibres which are in process of degeneration receive distinct differentiation. The degenerating nerve fibre is marked by the appearance of numerous black granula in its myelin sheath, which is evidently caused by a reaction of the osmic acid upon the decaying myelin. Normal fibres

receive an evenly grayish or brownish appearance. Degenerating nerve bundles are consequently distinctly differentiated from their surroundings by the numerous black granula which they contain.

But only during the process of degeneration does this reaction take place. When degeneration has been fully established the products of degeneration being absorbed the reaction fails, and it becomes just as difficult to distinguish degenerated fibres from normal ones as with the carmine, nigrosine and similar methods, especially where normal and degenerated fibres are closely intermingled. The animals must, therefore, be sacrificed before degeneration has been fully established. Experience has shown that three to seven weeks after the date of the experiment is the proper time to kill them.

A thorough knowledge of the method is required, however, in order to avoid mistakes. The presence of black granula is not always indicative of degeneration. Singer and Muenzer have shown that when a normal nerve, after being removed from the body, is pinched or otherwise injured the same black granula will be found at the point of lesion. They also stated the presence of black granula in regions where degeneration could not take place and where the specimen had apparently not been damaged. Whether this may be explained by the disintegrating influence of the hardening process itself cannot be said at present, but it proves that one must not push the diagnostic value of the reaction which may properly be called "*Marchi's and Algeri's reaction*" too far. Singer and Muenzer are fully aware of this fact, but they also state that by repeating the experiments one can avoid errors. The degenerating fibres will always present a large number of the granula, and the latter will be found in the same region if the experiment is repeated, while where the appearance of the granula is accidental their localization will vary in every individual case. It is the constancy and intensity of the reaction that gives it diagnostic value.

Working with the above described method Singer and Muenzer reached the conclusion that wherever a nerve fibre is degenerating it must show *Marchi* and *Algeri's* reaction. If the latter is not observed at the time, when according to experience it should be present, there is no real degeneration. Judging from this point of view, they came to the conclusion that after the

lesion of a peripheric nerve secondary degeneration occurs in the peripheric stump, but not in the central one. The alterations which take place in the latter they propose to call atrophy, when adults are concerned, or aplasia, when the newborn were operated upon.

Singer and Muenzer's statement is contradicted not only by Forel, but also by Darkschewitsch and Marinesco, although the latter authors applied the same method for their researches which S. and M. had used.

Darkschewitsch operated upon the facial, hypoglossal and sciatic nerves of full-grown guinea pigs. The nerves were cut or ligated or torn off of the medulla or spinal cord. The animals were killed six weeks after the date of the operation. The specimens were treated after Marchi's method. In spite of the variety of the experimental conditions the result was uniform: the roots of the cerebral motor nerves (VII., X., XII.), and both the anterior and sensory roots of the mixed nerve (sciatic) distinctly showed Marchi and Algeri's reaction in all cases operated upon.

To demonstrate the condition of the motor nerve cells as influenced by the injury D. repeated the experiments and hardened the specimens in Müller's fluid, staining the sections with carmine. He found a simple atrophy of the cells of the VII. and XII. centres. There was also a marked atrophy of the nerve cells in the anterior horn of the animals whose sciatic nerve had been operated upon, but it was less pronounced than in the other cases.

Marinesco cut the pneumogastric nerve of a half-grown rabbit (about two months old.) Three months after the operation Marchi and Algeri's reaction, viz., the appearance of numerous black granula, and chunks could be stated in the central stump of the nerve.

Mojieff (Moxaew) ligated the sciatic nerve of rabbits. The animals were killed three weeks after the experiment. M. then studied the changes both in the central and peripheric stumps, partly by treatment with osmic acid, partly by Marchi's method. He found distinct pathological changes in the ascending stump, but does not believe them to be identical with those found in the peripheric stump. While the fibres of the latter presented complete decay of the myelin sheaths, disappearance of the axis cylinders and proliferation of the nucle of Schwann's sheath, the anterior and posterior root fibres showed only the appearance of black granula



their structure remaining normal otherwise. (No changes of the axis cylinders, no proliferation of the nuclei of Schwann's sheaths.) The appearance of the black granula, stated also in the roots of the healthy side, was pathological on the side of the operation by their larger quantity and larger size.

It can be seen from the above review of the literature that the opinions on the nature of the retrogressive process occurring in the central stump of a severed motor nerve are still divided. Thinking that further researches might clear the matter up, I resolved to add some casuistic material by aid of the experimental method.

I intended originally to study the condition of nerve fibres only, for which purpose I chose Marchi and Algeri's method, as the arguments put forth by these authors in defense of it seemed rather convincing to me. In the further course of my investigations I employed Nissl's method in order to study particularly the structure of the cells as influenced by the lesions created.

In selecting the physiologic anatomical material it first struck me that to my knowledge no author had used the dorsal nerves for the basis of his experiments, although many factors speak in favor of this choice. In the first place the area of the distribution of most dorsal nerves is well defined and anastomoses of one nerve with branches of another are exceptional; consequently the fibres of the central stump correspond to those of the peripheric one. In the sciatic and brachial plexus, however, anastomoses are so numerous that a proper comparison of the central with the peripheric stump cannot be made, the former containing quite other fibres than the latter. This is illustrated by the fact that according to Gowers the median and musculo spiral nerves derive their fibres from the same roots, viz., the fifth, sixth, seventh and eighth cervical. (To the formation of the musculo spiral nerve the fourth cervical root also contributes).

A similar proof is furnished by the interesting case which Müller reports. Here a lesion of the lowest two cervical roots caused a paralysis of all the motor and sensory nerves for the forearm and hand, while the functions of the nerves for the arm and shoulder remained perfectly normal. Or in other words: All branches of the cutan. brach. med., ulnar, median and musculo-spiral nerves which supply the forearm with

## PLATE I.

FIG. 1. Dorsal portion (lower part) of a normal spinal cord of a young cat.

The illustration is a photograph showing the distribution of the ganglionic cells:—

Med = cells of the medial zone (Waldeyer's Mittelzellen).

Later. = group of the lateral horn.

Post. = group at the base of the posterior horn.

Magnifying power 47 lin. Vesuvine stain.

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FIG. 2. Photograph. Normal anterior horn cells.

Magnifying power 260. Vesuvine stain.

# PLATE I.

FIG. 1.

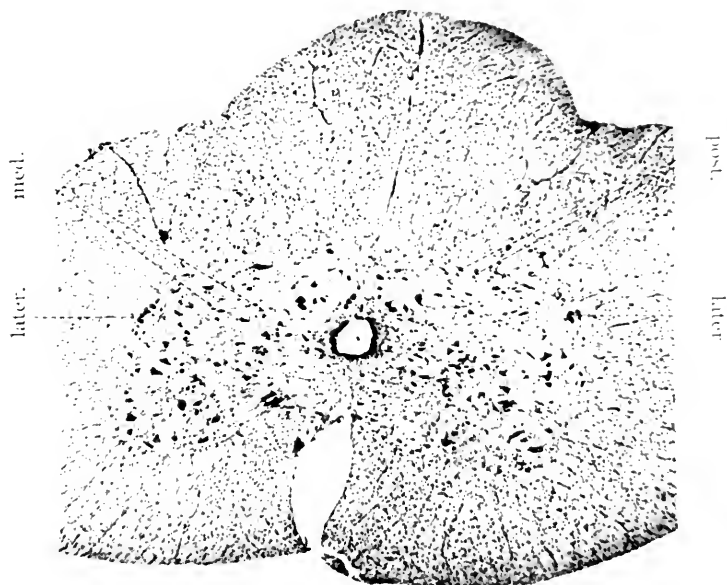


FIG. 2.

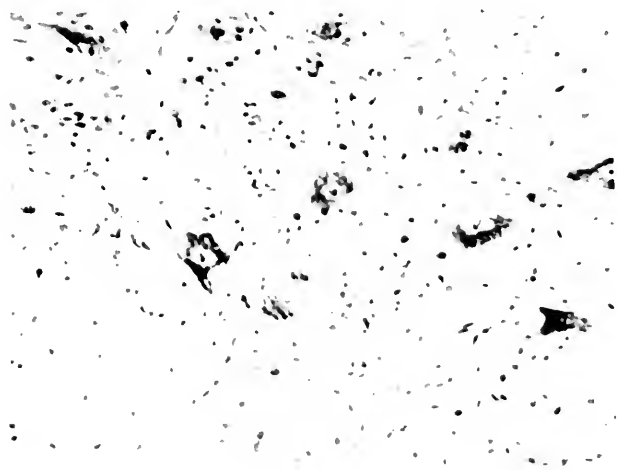


Fig. 1 reduced to  $\frac{1}{17}$  of size of original plate, the magnifying power is therefore not 47 but 34 lin.

Fig. 2 reduced  $\frac{1}{16}$ , accordingly magnified 268 lin., not 290 as on original plate.



motor and sensory nerve fibres were paralyzed, while those branches of the same nerves which distribute their fibres in the arm remained perfectly normal.

The facts mentioned heretofore also bear reference to the distribution of the motor (and sensory) nuclei in the cord. It seems probable a priori that one dorsal segment functionally corresponds to the one dorsal nerve, while one segment of the cervical or lumbar enlargements corresponds to several peripheric nerves, these latter deriving only part of their fibres from this one segment.

What further complicates the conditions in the case of the sciatic or brachial plexus is that the lumbar and cervical enlargements having a more complex function, possess a larger number of association fibres and nerve elements which are in no direct connection with the nerves.

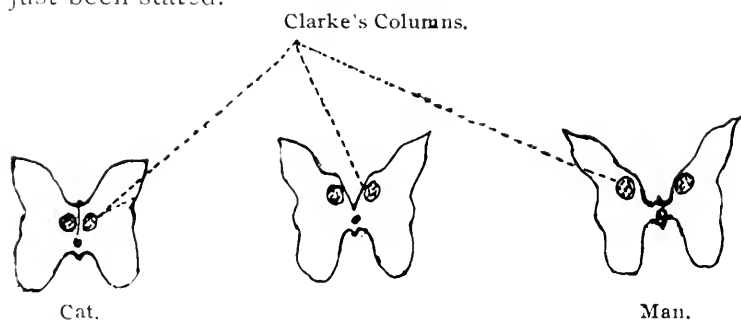
Having all these points in mind, I decided to select the dorsal nerves as a ground work for my experiments. The nerves operated upon were the eighth, ninth or tenth dorsal. In one case it was one of the lowest, but I did not mark exactly which. In all cases the experiments were performed on nerves of the left side. I chose cats for my experiments as the proverbial nine lives of those animals gave hope that most of them would survive the injury inflicted. A further advantage of experimenting upon cats is that the wounds heal rapidly, although not necessarily per primam intentionem. It is very important, of course, to proceed strictly aseptically, but although I observed the strictest asepsis during the operation I found it very difficult to keep up an aseptic condition of the wound later on. The bandages were usually sooner or later gnawed through or in some way torn off. So after several discouraging experiences with antiseptic bandages I finally decided to leave them off altogether. I sewed the wound up and removed the sutures twenty-four hours afterwards. If pus had formed in the meantime it would come out as soon as the sutures were removed. Under such circumstances I would sometimes reunite the wound by one or two sutures, but leave it sufficiently open to allow of a free discharge of the pus.

The age of the animals experimented upon varied between four and five weeks. As it was my intention to especially study the ascending changes of motor fibres, I began the series of experiments with isolated section of the anterior root of a dorsal nerve.

Before I enter upon the report of my observations I wish to mention a few anatomical facts relating to the structure of the dorsal portion of the cord. In the first place it must be stated that the dorsal cord of the cat differs in several respects from that of the human cord, namely, in the shape of the gray commissure and in the location both of the central canal and of Clarke's columns. The gray commissure of the cat is comparatively much larger in a dorso-ventrad direction than that of man. In the human cord the central canal occupies the middle of the gray commissure, while in the cat it lies ventrad quite near its anterior (ventral) border, so that by far the largest part of the commissure is situated dorsad from the central canal. The median line of this enlarged dorsal portion of the commissure is marked by a sort of septum at both sides of which near the central canal Clarke's columns are located, while in man these cell groups are situated at the median border of the base of the posterior horn dorsad and somewhat laterally to the gray commissure.

This difference of structure is not as fundamental as would appear at the first sight, and if we imagine the dorsal part of the cat's gray commissure divided in the median line by a force which would crowd the severed portions laterally, the effect would be to equalize not only the width of the commissure, but also the location of the central canal and of Clarke's columns with these of man.

The following diagrams will best illustrate what has just been stated.



Another point of difference is given in the shape of the posterior columns, which in the cat appear larger in a dorso-ventrad direction, the posterior (dorsal) outline being more convex than it is in man. This might be

explained by the increased width of the gray commissure which would naturally displace the posterior columns in a dorsad direction.

In other respects the dorsal portion of the cord of the cat is quite similar in structure to that of man; but there are some points in which it differs from that of the lumbar and cervical portions of the cord.

I wish to call particular attention to the distribution of the ganglionic cells.

In the first place it needs mentioning that the division into groups is much less pronounced than in the lumbar and cervical portions of the cord. The best defined group is that of Clarke's columns, situated, as I have explained, dorsad from the central canal at both sides of the median line. Its cells are on the average smaller than the multipolar cells of the anterior horn, although varying quite considerably in size. They are frequently grouped in an oval or circle surrounding a stratum of nerve fibres. Occasionally one meets with a large solitary cell (mostly multipolar) situated exactly in the median line of the dorsal part of the gray commissure close by the central canal. The cell body of the cells of Clarke's columns usually presents itself in an oval or round shape with a large nucleus whose form varies with that of the cell. The number of processes is usually small.

Another rather well-defined group is that of the lateral horn. The latter is much less conspicuous than it is in the lumbar portion of the cord. It is marked only by a slight convexity of the lateral border of the gray matter. In many sections, however, triangular processes are seen projecting towards the lateral columns. The cells of said group are small, mostly of pyramidal or spindle, sometimes, as for instance, in Fig. 1, Plate I, of round shape. They are frequently crowded into nests arranged in a chain like manner. The triangular processes mentioned are usually occupied by a crowded nest of cells. In location this group corresponds to the postero-lateral group of the lumbar enlargement, but its territory is much smaller. In the lumbar enlargement it is predominantly composed of large multipolar cells, while in the lateral horn of the dorsal cord large cells are rarely met with. It must further be stated that the group of the lateral horn of the dorsal cord shows off indistinctly against the surrounding parts, its cells becoming intermixed with those of the adjoining portions of the gray matter.

The antero-lateral, antero-internal and central groups are also represented in the dorsal portion of the cord, that is, we find large multipolar cells in the corresponding regions, but the arrangement varies so much that anatomical distinction of definite groups is hardly possible.

In that zone, which is situated between the base of the anterior and that of the posterior horn, and which might properly be called the medial zone, we find also numerous cells. Most of them are small or of medium size, of oval, spindle or pyramid shape; occasionally large multipolar cells are also seen. Quite frequently the smaller cells group in nests at the side of the central canal.

It but remains to mention one group of cells which I found to be quite constant. These cells are located between the bundles of nerve fibres, which, originating from the antero-lateral part of the posterior columns (mostly as a continuation of the median bundles of the posterior roots), pass in a curve through the base of the posterior horn towards the lateral columns.

The cells mentioned are usually very small, long-stretched, and their longitudinal axis coincides with the direction of said nerve bundles, between which they are arranged in chain-like tracts. Laterally these cells join the group situated at the lateral border of the gray substance between the lateral and posterior horns, which Bechterew calls the lateral group of the posterior horn. The cells of this latter group are, as a rule, much larger than those mentioned before, and mostly polygonal.

Having mentioned already that my first experiment consisted in isolated section of the anterior root of a dorsal nerve, I shall now give a description of the case:

I.—Cat, 33 days old. Section of the anterior root of one of the lowest dorsal nerves of the left side (which nerve it was, cannot be exactly stated).

Experiment performed under ether narcosis.

Longitudinal incision over process. spinos. of vertebrae about one and one-half inch in length. Severing of insertion of muscles from the spinal column.

Resection of a piece of the covering of the vertebral canal by making two longitudinal sections with the saw about two mm. laterally from the process. spinos. and each measuring about one inch in length. Removal of the loosened piece with the bone forceps. I preferred this manner of procedure to using the bone forceps

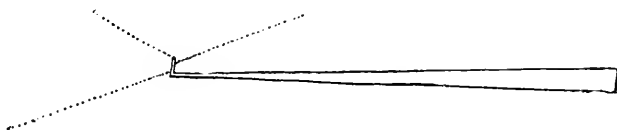


exclusively, as in the latter case the bleeding is much more profuse.

The vertebral canal now being opened, I waited until the bleeding had entirely ceased, then removed the blood coagula partly by irrigation with sterilized water and partly by gently withdrawing them with the forceps. The roots were then exposed by removing the fat tissue that covers them. Some slight bleeding occurred again which ceased very soon. I then removed the blood coagula in the manner mentioned and proceeded to the section of the root. This was done with a small slender knife devised especially for that purpose. The following drawing will better explain this instrument than a lengthy description.

Blunt point of knife.

Blunt back of knife.



Cutting side of knife.

The knife was introduced between the two roots, after which the anterior root was pressed ventrad against the bone and cut through. The wound was then irrigated and sutured.

The animal was quite lively after the effects of the narcosis had passed over. At no time did it show any symptoms of shock or injury to the spinal cord. The gait was perfectly normal. The wound healed by secondary reunion. Four weeks after the date of the experiment the cat was killed by desanguination.

Examination revealed a cartilage-like covering of the vertebral canal at the locus of operation. After removal of this, covering tracts of a tissue similar to "jelly" in appearance and consistency were observed which formed adhesions between the dura mater of the cord and the vertebrae. These adhesions were easily removed without damage to the membranes which appeared normal.

The spinal ganglion that corresponded to the operated root was found embedded in a jelly-like mass of tissue, from which it was not possible to separate it. The posterior root seemed macroscopically normal. The anterior root which passed directly into the jelly-like mass just

mentioned, was found transformed into a thick bundle of a soft, jelly-like tissue.

A piece of the spinal cord measuring about one and one-half inches in length and containing the segment which corresponded to the severed root was put in Müller's fluid for the purpose of preparing it for handling by Marchi and Algeri's method. I did not find it necessary to cut it into smaller pieces, as the thickness of the cat's spinal cord is only about one-half centimetre in this region. The peripheric nerve was also put into Müller's fluid. Both spinal cord and peripheric nerve were afterwards treated according to the directions of Marchi and Algeri, whose method I have described in some detail on page 606.

#### MICROSCOPICAL EXAMINATION.

##### *1. Of the nerve:*

It shows typical degeneration, the medullary sheaths of its fibres being impregnated with a large number of black granula and chunks. For comparison the corresponding nerve of the other side and other dorsal nerves were used.

##### *2. Of the spinal cord:*

A continuous series of about 450 transversal sections was made, each section varying in thickness from one-fortieth to one-eightieth mm. I shall now first describe the appearance of normal specimens which were treated after Marchi's method.

The white substance has microscopically a dark gray or brownish, the gray substance a light yellow or gray tinge. Microscopically the transverse sections of nerve fibres show a rather distinct differentiation of axis cylinder and medullary sheath, the former being of a dark greenish or black, the latter of a light gray color. On longitudinal sections of fibre bundles the axis cylinders appear as dark lines which show off rather distinctly against the lighter colored medullary sheaths. In the gray substance this differentiation is much less pronounced. (The appearance presented by the ganglionic cells I shall have occasion to describe later on). Here and there black granula or masses are seen scattered between the nerve fibres; their arrangement is quite irregular; occasionally, however, one will be found to correspond to the transversal section of a nerve fibre. They are chiefly located in the white substance; in the

gray matter they are rarely met with. The localities in which these masses and granula are most frequently observed are the direct horizontal bundles of the posterior and anterior roots.

Let us now take the transverse sections of the level which corresponds to the severed anterior root.

#### I. WHITE SUBSTANCE.

The posterior column of the left side is much narrower than that of the right side. This lessening is due to a lessening of the entering posterior rootlets and of their continuation into the zone of vertical fibres situated laterally from the median border of the left posterior horn (medial fascicle of the posterior roots. Bechterew). The medullary sheaths have disappeared to a great extent, and the axis cylinders are crowded together so that they can hardly be distinguished from each other. Aside from this a large number of black granula are seen in said zone, viz., along the median border of the left posterior horn, while on the right side only a few are observed in this area. The presence of the few granula on the right side must be considered as accidental, while on the left side their large quantity and constancy is indicative of degeneration. This degeneration of the left posterior root was, of course, uncalled for, probably it was caused by the proliferation of interstitial tissue in which the posterior root and spinal ganglion were found imbedded.

The anterior roots of the left side present a considerable number of black granula and masses, but these are just as numerous in the anterior roots on the right side. Indeed, in spite of a careful examination of a large number of sections I could not notice any difference between the anterior roots of the right and those of the left side. If there was any degeneration I could not prove it by Marchi's reaction.

The antero-lateral columns of the left side are exactly like those of the right side. No signs of degeneration can be discovered.

#### II. GRAY SUBSTANCE.

As I have explained, the purpose of Marchi and Algeri's method is to produce a reaction which enables us to recognize the process of degeneration in nerve

## PLATE II.

Illustrates the retrogressive cell changes observed four weeks after the section of the anterior root of a dorsal nerve of a young cat. Treatment of the specimen after Marchi's method.

FIG. 1. Water color.

Magnifying power 47 lin.

FIG. 2. Water color.

Magnifying power 320 lin. Showing various stages of the retrogressive changes.

# PLATE II.

FIG. 1.

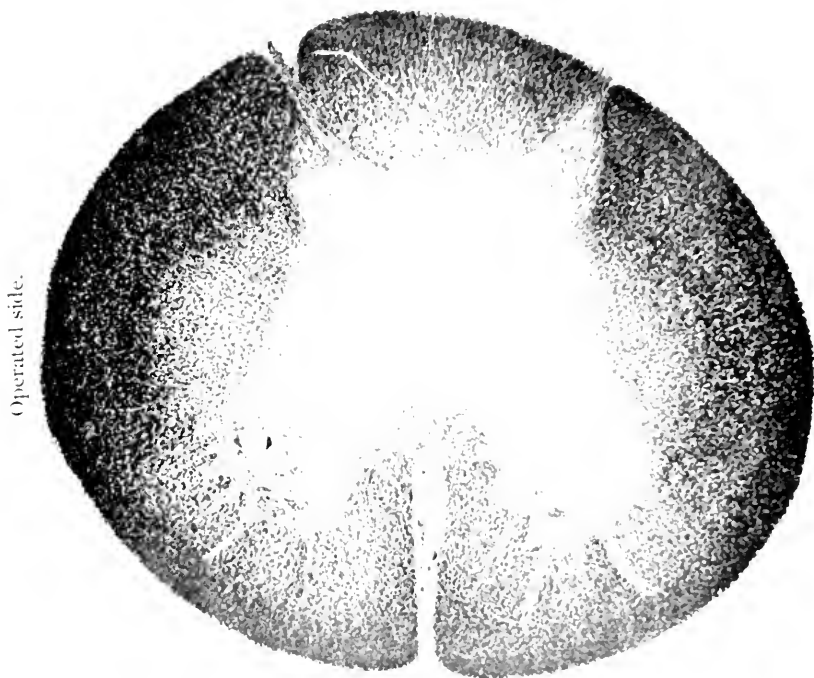


FIG. 2.



Fig. 1 reduced to  $\frac{1}{17}$  of size of original illustration, the magnifying power is accordingly not 17 but 28 lin.  
In Fig. 2 the reduction is very slight.



fibres: it has not been used to my knowledge for the recognition of cell changes. Indeed, Darkschewitsch, who, besides studying the degeneration of nerve fibres with Marchi's method, who wanted to get information on the condition of nerve cells as influenced by injuries of peripheric nerves, repeated his experiments, and after hardening the specimens in the usual way with Müller's fluid, stained the sections with carmine. That the method was not meant for defining the condition of cells is also clear from the fact that no author even mentions what appearance the ganglionic cells of "Marchi-specimens" show.

My observations, however, induce me to pay special attention to this point.

In sections of normal specimens that were treated after Marchi's method the cell, have a shrunken appearance, they are so badly differentiated from the surrounding tissue that with weak magnifying power (50 lin.) at first no cells whatever are distinguished. By looking more closely one will observe spots of irregular shape which are somewhat darker than the surroundings. With a stronger magnifying power (300) these spots can be identified as cells, which, however, show quite a rudimentary shrunken chunk-like appearance. Seldom can a differentiation of nucleus and nucleolus be made: the processes are sometimes present, but are also shrunken and rudimentary, more frequently, however, they cannot be recognized altogether, the cell being represented as a structureless mass. After having been examined with a strong magnifying power the cells are also recognized with a weak power, or, better said, they can be guessed at.

It is evident, therefore, that normal ganglionic cells receive a very poor differentiation by Marchi's method.

Let us see how the ganglionic cells which correspond to the injured anterior root present themselves in our specimen:

In examining the left anterior horn at the level of the injured root we see a considerable number of large cells with well-defined outline, distinctly differentiated nucleus and nucleolus and more or less developed processes. In comparing them with normal cells stained after Nissl's method, we find them considerably enlarged. This increase in size is due to a swelling which manifests itself by their rounded shape, the outlines of the cell body between the processes being convex instead of

concave as in the normal cells. The swelling also involves the nucleus and the processes which latter mostly have a thick clumsy appearance. In many cells the processes look as if broken or fallen off, representing short thick stumps.

As I have mentioned, the outline is well defined in most of the cells, but one finds others in which it is blurred, so that the cell body can be properly compared to a half dissolved piece of celloidin or gelatin.

Some cells of the latter type will still present a well differentiated nucleus, in others the nucleus seems to have dissipated itself in the cell body altogether, although the nucleolus, which evidently preserves itself longest, may still be distinguished.

On the other side, but less frequently than the type just described, are seen cells with a sharp outline and rather well-developed processes, but without nucleus and nucleolus.

Occasionally we find a cell in which part of the cell-protoplasm has another appearance than the rest of the cell body; it looks as if a foreign body was located in the cell.

These various cell types represent various stages of the same pathological process, namely, of a homogeneous degeneration. This degeneration begins in the cell body (as I shall demonstrate later on) and then gradually involves the other parts of the cell, usually first the processes, then the nucleus, and finally the nucleolus, sometimes, however, first the nucleus and then the processes.

The earlier stages of the degeneration are marked by swelling and by distinct differentiation of the degenerating parts with Marchi's method. In later stages a sort of liquefaction evidently occurs by which the differentiation becomes lost in such a manner that single parts of the nerve element may still retain distinct differentiation and seemingly normal structure when the others are destroyed already.

In location and shape the described degenerated cells correspond to the large multipolar cells of the anterior horn, which are considered to have motor functions. The degenerated cells are most numerous in the left anterior horn, but also met with in the right one, the proportion being more or less three (side of operation) to one (other side). They are distributed over all groups of the anterior horns.



We find then, that the section of the anterior root of a dorsal nerve in a young cat produced within four weeks a degeneration of the large multipolar cells of the anterior horns, cells to which we ascribe motor functions. This degeneration must be called a homogeneous degeneration from reasons which I shall explain later. The beginning stages of said degeneration are characterized by a swelling of all parts of the cell and by their distinct differentiation with Marchi's method in contrast with normal cells which receive a very poor differentiation and shrunken appearance with said method.

The observations published above seemed to me so suggestive that I resolved to endeavor to elucidate the matter by further investigations. It had occurred to me that the condition of "motor" ganglionic cells as influenced by injuries of motor fibres had never been studied, yet at early periods after the experiment, but always at a time when the retrogressive process had presumably reached its final stage. It then occurred to me that possibly the described "pseudohypertrophic" condition of the anterior horn cells represented an early stage of degeneration which finally would lead to atrophy or complete disappearance of the cells. To determine whether my conjecture was justified, I decided to start a new series of experiments, to kill the animals at different periods after the experiment and to study the condition of the ganglionic cells with a method by which presumably the finest details of the cell structure could be discerned. Nissl's stain seemed to be the most fit and reliable for that purpose. Nissl's publications on the cell alterations of the VII. nucleus following the injuries of the VII. nerve were not known to me at that time; indeed, while collecting the literature on the subject in question I sought for his first publication regarding the facial nerve, which I found cited by various authors, but did not discover it until lately. I can say, therefore, that the following investigations were made quite independently, without knowledge of Nissl's observations. I am indebted to Dr. Spitzka for having called my attention to Nissl's second publication concerning the facial nerve, which indirectly led me to the discovery of his first publication. Having made my investigations without knowledge of Nissl's researches, it was so much more gratifying to me, that his results concerning the facial nerve coincide in the essential points with those which I obtained by operating upon the dorsal and intercostal nerves.

I am sorry not to have used Nissl's improved method for my researches as its publication had also escaped my attention. I employed his old method as described in the second edition of Obersteiner's "*Anleitung zum Studium der nervösen Central-Organen*," but with certain modifications which will be seen from the following description:

Hardening of the fresh specimen in ninety-five per cent. alcohol from about one to two weeks, then in absolute alcohol for a further week, subsequently drenching with xylol for one day, then with solutions of paraffine in xylol of increasing concentration (until saturated) for about three or four days; finally drenching in melted paraffine at a temperature not exceeding the melting point, that is, at about 110° F. for two hours.

The sections were stained upon the slides in the following manner:

Fastening of the sections upon the slides by means of a mixture of rapidly filtered egg albumen and glycerine at equal parts (after P. Mayer). Smoothing of the sections with a moistened camel's hair-brush until they are quite even and adhere so firmly to the glass that pressure with the brush cannot displace them any more. The superfluous fixing fluid thus pressed out is wiped off from the slide. This done, a few drops of xylol poured over the section quickly dissolve the paraffine: this process is repeated, after the xylol first put on has been wiped off, until every trace of paraffine is out of the section. In the same manner the xylol is washed out of the section with absolute alcohol. Presently the slide is first transferred into water or directly into the staining fluid, a concentrated aqueous solution of vesuvine, wherein it is kept for about one hour (I made this modification, because by heating the sections shrivel). The superfluous staining fluid is subsequently washed out by putting the slide into water for a few seconds, keeping it longer in water does no harm. The water having been wiped off from the slide a few drops of alcohol are poured over the sections. The alcohol must be kept on long enough to abstract all water out of the sections which it does in a few seconds; keeping much longer will spoil the stain. The next step is to wipe off the alcohol and pour oil of cloves on the sections. The effect of the differentiation in the oil of cloves can now be watched directly under the microscope, which is a great advantage in my modification. (The mounting of the

sections is best done by Nissl's benzine-colophonium method, as later experiences have shown me.)

Comparison of my "paraffine" sections with sections of a specimen which did not undergo the paraffine drenching, but was cut right after the hardening in alcohol, convinced me that the process of paraffine drenching did not damage the structure of the cells. I also compared the cell stain of my "paraffine" sections with the drawings of nerve cells with Alzheimer, who employed Nissl's improved methyl blue method gives in his monograph mentioned at the end of this article. This comparison proved that the cell structure as presented in these drawings does not differ from that which I found in my "paraffine" sections. I feel justified, therefore, in concluding that my modifications did not impair the results of the stain.

It would lead too far to give a detailed description of the structure of the various types of ganglionic cells as differentiated by Nissl's stain. Nissl himself has described them so accurately that I need only call attention to his numerous publications on that subject (Vide literary register at the end of this article), I shall but mention the essential points:

Nissl's stain reveals the existence of two parts in the substance of the cell body, one of which accepts the stain "chromatophile part of the substance," while the other remains unstained. Of the chromatophile part of the substance one portion becomes intensely stained, the other but slightly.

The intensely stained part is represented by the "corpuseles" of various shapes. In longitudinal cells spindle-shaped and stripe-like corpuseles prevail, in others, round, oval, angular and irregular forms are chiefly found. In many cells—and especially is this the case in the large multipolar cells of the anterior horns—these corpuseles are arranged in rows which in the vicinity of the nucleus have a tendency to run parallel with the outline of the latter, while in the peripheric part of the cell they mostly run parallel with the outline of the latter. In longitudinal cells they are usually more or less parallel with the longitudinal axis of the cell. In the processes the corpuseles appear in the shape of spindles, long-stretched pyramids or stripes, the longitudinal axis of which is directed parallel with that of the outline of the process.

To many cells, for instance to the large multipolar

cells of the anterior horns, this arrangement gives an appearance similar to that of the tiger hide (Nissl. [See Plate I., Fig. 2].

The slightly stained part of the substance is more delicately organized and shows itself in the form of minute grains, finest threads, chains of minute grains, net works of finest threads, etc.

I shall now return to the subject in question and enumerate the experiments performed.

As mentioned before, the age of the cats upon which I experimented varied between four and five weeks:

Experiment 1.—Section of the anterior root of the left ninth dorsal nerve. Animal killed three days after operation.

Experiment 2.—Section of the anterior and posterior roots of the left eighth dorsal nerve. Animal killed six days after operation.

Experiment 3.—Section of the anterior root of the left eighth dorsal nerve. Animal killed fourteen days after operation.

Experiment 4.—Section of the left tenth intercostal nerve. Animal killed three days after operation.

Experiment 5.—Section of the left ninth intercostal nerve. Animal killed six days after operation.

Experiment 6.—Section of the left tenth intercostal nerve. Animal killed fourteen days after operation.

The intercostal nerves were severed peripherically from the dorsal branch which supplies the muscles of the back. Experiments 2 and 6 were performed upon the same animal. Experiments 3 and 4 were also performed upon one animal. For experiments 1 and 5 two separate animals were used.

In cases 1, 3 and 6 changes were found which could hardly be the result simply of the lesion intended, but were evidently partly caused by an abnormal inflammatory proliferation of the neuroglia elements. In one case (VI.) a kind of area of softening formed. In case IV. the series of sections was not complete and the stain deficient.

The clearest and most distinct results were those of Experiment II and V. I shall, therefore, first report these, and then mention as much of the others as may serve to corroborate the observations made in cases II and V.

EXPERIMENT II.—Section of the anterior and posterior roots of the left eighth dorsal nerve. Animal killed six days after the operation.

After the operation the animal showed some ataxy in the movements of the hind legs and a tendency to fall over when making a sharp turn. The symptoms disappeared within a few hours; afterwards the animal was very lively, played with the other cats, ran and jumped with much grace and skill, etc. The wound healed partly by primary and partly by secondary reunion. The cat was killed with chloroform six days after the date of the experiment.

Examination of the region of operation revealed that both roots had been entirely severed, and that the ends of the peripheric stump were so far removed from those of the central one as to surely exclude the possibility of a reunion.

#### MICROSCOPICAL EXAMINATION.

As with Nissl's stain the outlines of the gray substance became very indistinctly marked, some of the sections were stained with carmine. These sections show that the left posterior column is larger than the right one, both in dorso-ventrad and frontal direction. The region which corresponds to the tract of Lissauer seems also enlarged (although on account of the alcohol hardening which renders the structure of the nerve fibres very poorly, its recognition from the adjoining zones becomes very difficult).

This increase of volume of the left posterior column is evidently due to beginning degeneration of the posterior root zones. It seems natural to assume that in the beginning of the degeneration a swelling of the medullary sheaths occurs which would satisfactorily explain the increased volume of the left posterior column.

The antero-lateral columns are equal on both sides.

There is an asymmetry of the gray matter. The left posterior horn looks as if compressed in a radial (from apex towards central canal) direction, having a hemispherical shape. Its apex is much further removed from the dorsal periphery of the cord than that of the right one. Whether a difference in volume exists between the posterior horns, it is hard to decide on account of the asymmetry mentioned.

The left anterior horn is longer (ventro dorsal), but

## PLATE III.

(Exp. No. II.) Illustrating the alterations which follow the section of the anterior (and posterior) roots of a dorsal nerve within six days.

FIG. 1. Photograph showing the dimensions of the gray and white matter.

Magnifying power 13 lin. Carmine stain.

FIG. 2. Photograph of a normal anterior horn.

Magnifying power 111 lin. Vesuvine stain.

FIG. 3. Photograph of the anterior horn, the cells of which show homogeneous degeneration.

Magnifying power 111 lin. Vesuvine stain.

*n* is a normal cell, the others are degenerated.

FIG. 4. Water color, showing various stages of the homogeneous degeneration.

Magnifying power 320 lin. Vesuvine stain.

*a.* Cells presenting a circumscribed area of homogeneous degeneration of the cell body, the periphery of which has still a distinctly granular structure. Nucleus apparently normal in one cell, in the other only the nucleolus is still preserved.

*b.* Cells in which the nucleus has partly left the cell body.

*c.* Swollen cells, in two of which the nucleus is becoming dissolved in the cell body.

*d.* Late stages of the homogeneous degeneration, two cells represented only by structureless chunks.

# PLATE III.

FIG. 1.

Operated  
side.



FIG. 2.

FIG. 3.



FIG. 4.



Fig. 1 reduced to  $\frac{1}{3}$  of size of original illustration.  
 Figs. 2 and 3 reduced to  $\frac{1}{10}$  of size of original illustration.  
 Fig. 4 reduced to  $\frac{1}{4}$  of size of original illustration.  
 The magnifying power is accordingly—  
 For Fig. 1 9 lin. (not 13).  
 For Figs. 2 and 3 77 lin. (not 111).  
 For Fig. 4 256 lin. (not 320).





narrower (frontal) than the right one. This asymmetry seems to be caused by distortion; there does not seem to exist a difference in volume between the two anterior horns, although also here it is rather difficult to make a decision on that point.

No signs of inflammation are noticed anywhere. There is an increase of neuroglia elements in the left lateral horn, part of the cells of which are nearly buried under granular cells. Aside from this a proliferation of neuroglia elements is not noticed, especially not in the anterior horns.

There is a considerable lessening of the number of cells of the left column of Clarke and of the small cells of the left lateral horn. A large part of the remaining cells of these groups are shrunken, intensely stained and otherwise altered. Changes of the same nature are seen in cells of the zone situated between the basis of the left anterior and posterior horns (medial zone) and in many of the cells located at the base of the left posterior horn, a description of which group I gave in a previous section.

On the right side changes of the nature just mentioned are also observed in cells of Clarke's column, of the middle zone (between bases of anterior and posterior horns) and of the group at the base of the posterior horn.

The most conspicuous alterations of structure were found in the

#### LARGE MULTIPOLAR CELLS OF THE LEFT ANTERIOR HORN.

The most important features of the changes are succinctly stated in the following summary:

1. The differentiation of the chromatophile and chromotophobe parts of the substance of the cell body is lost. The cell-body has a homogeneous appearance.
2. The homogeneous cells have a pale color.
3. The homogeneous transformation is usually, although not necessarily accompanied by a swelling of the affected parts.
4. The processes undergo the same transformation as the cell-body.
5. The nucleus and nucleolus also undergo retrogressive changes; they finally become absorbed in the cell-body or the nucleus leaves the cell-body and probably becomes absorbed outside of the latter.

6. The degeneration evidently begins in a circumscribed part of the substance of the cell-body; the degenerated area then increases involving gradually the other parts of the cell.

7. The final result of the homogeneous degeneration is probably a liquefaction and absorption by endosmosis or by means of granular cells, although the degeneration may be arrested before this final stage is reached.

8. The cells thus affected are the large multipolar cells of the left anterior horn. A small number of degenerated cells is also found in the right anterior horn.

9. The said degeneration is caused by the section of the anterior root of a dorsal nerve.

In many of the diseased cells the whole cell-body including the processes, has a perfectly homogeneous appearance, in others the centre is homogeneous while the peripheric part shows still one or several distinct rows of intensely stained "corpuscles" (a name which Nissl proposes instead of granula).

Friedmann first promulgated the view that the homogeneous degeneration which he calls "homogeneous swelling" begins in a circumscribed part of the cell-body and progresses centrifugally. I can only affirm the correctness of this view by my observations. In many of the affected cells indeed only a small circumscribed area of the cell-body is homogeneous, the remaining part of the cell including the processes and nucleus having the characteristic granulated or striped appearance which Nissl's stain gives them.

In a large number of the affected cells the nucleus shows apparently normal structure, in others it has a peculiarly light and shining appearance, the minute granula which it normally contains being scarce and the outlines being sharply defined, better than in normal cells where they are frequently hidden from view by the intensely stained "corpuscles" of the cell protoplasm covering them. Most frequently the nucleus is dislocated towards the periphery, bulging the outline of the cell-body out so as to look like a projection attached to the cell. In several cells I could state an actual "luxation" of the nucleus, the greater part of it lying outside of the cell-body. Probably the nucleus becomes entirely eliminated from the cell-body in this manner and is absorbed outside of it. In many cells, however, only a nucleolus is seen or the outlines of the nucleus are so indistinct, blurred that they can hardly be recognized. In these

cases the nucleus is probably absorbed by the cell-body itself. The nucleolus preserves itself longest.

I have mentioned that in many of the degenerating cells the processes show still a normal structure when the cellbody or part of it is already homogeneous; in others they are swollen and look homogeneous, or they have fallen off, only short stumps being left, or they have disappeared altogether.

Cells without processes but with apparently normal nucleus are seen. On the other side cells with apparently normal processes but without nucleus or without nucleus + nucleolus are met with.

Finally one finds homogeneous chunks in which every trace of the nucleus, nucleolus and processes has disappeared. These latter formations, of course, represent advanced stages of the process of degeneration.

To sum up, a large number of the large multipolar cells of the left anterior horn and some of the right anterior horn have undergone retrogressive changes which are analogous to what Friedmann calls homogeneous swelling or homogeneous degeneration (*homogene Schwellung, glasige homogene Entartung*). The degeneration begins in a circumscribed part of the substance of the cell-body and progresses centrifugally; the processes usually are next involved, earlier than the nucleus and nucleolus, but there is no doubt that quite frequently the nucleus degenerates earlier than the processes. As in many cells the outlines of the cell-body are quite blurred, it seems very probable that in the later stages a liquefaction takes place which finally leads to absorption of the cell. One manner of absorption is certainly the being eaten up by granular cells. I could see this in the case of the cat on which section of the anterior root of a dorsal nerve had been performed, and which was killed three days after the experiment. Here I also observed homogeneous degeneration of multipolar cells of the anterior horn and could make out that many of the degenerated cells had quite blurred outlines and were quite "buried" beneath granular cells.

If we compare the cell changes mentioned with those which were found in the spinal cord treated after Marchi's method (section of anterior root of a dorsal nerve, vide page 620) we find that there is a considerable analogy. The swelling of the cells was less marked in the Nissl specimen, but this may be explained by the direct and therefore stronger action of the alcohol, while

in the Marchi specimen hardening with Müller's fluid and osmic acid preceded the alcohol treatment. Otherwise the alterations were so similar that in my opinion the process must have been the same in both cases.

Nissl has studied the retrogressive changes which tearing off of the VII nerve from the base of the brain caused to take place in the cells of the VII nucleus. He examined the condition of the cells 1, 2, 3, 4, 6, etc., days after the experiment. His description coincides with mine in the essential points, only he speaks of a finely dusted, not of a homogeneous appearance of the diseased parts. Considering that I did not study the process of degeneration at as early stages as he did, I could not give as accurate a statement of the retrogressive changes. Where the degeneration was still in the beginning, I could also frequently observe a finely granulated or dusted appearance of the affected parts. It may be also that the modification which I used gave somewhat less accurate results than Nissl's new methylen blue method.

In the case of amputation which Kahler and Pick have described (*vide* page 603 of this article) the alterations found in the cells of the anterior horn of the lumbar portion of the cord were evidently of the same nature as those which I described as homogeneous degeneration. I shall mention some details of Kahler and Pick's report which clearly demonstrate this: The affected cells are decidedly enlarged—they appear paler and much more evenly stained—they have a homogeneous colloid appearance—the nuclei with very distinct nucleoli are located quite peripherically and in some sections appear like a projection attached to the cell."

That which makes Kahler and Pick's case especially interesting is that the patient died at as late a period as eighteen years after the amputation. This proves that the homogeneous degeneration can arrest itself at a certain stage and that the cells can preserve themselves for years in the changed condition. It follows that the homogeneous degeneration of a "motor" cell as caused by severance of the continuity of the motor fibre which originates from it does not necessarily lead to complete destruction of the cell even when the continuity of the fibre remains unrestored.

This, furthermore, suggests that in the beginning of homogeneous degeneration the "motor" cell may still be capable of "recovery," if the function of the motor fibre can be restored.

The experimental researches of Lubinow on the regeneration of nerve cells in the cerebral cortex, which had undergone retrogressive changes under the influence of the disturbances of nutrition caused by starvation are very illustrative in that regard. L. could observe the regeneration of cells, the cell-body of which had nearly completely disappeared; the remaining protoplasm accumulates around the nucleus in shape of a narrow border, which becoming broader and broader begins to give off "sprouts" which evidently are the beginning of newly-formed processes.

EXPERIMENT V.—Section of the left ninth intercostal nerve near, and peripherically from the dorsal branch which supplies the muscles of the back. Animal killed six days after the experiment.

Operation performed under ether narcosis. The peripheric stump was loosened somewhat from the underlying tissue, otherwise the stumps were not dislocated.

The animal was very lively and jolly as soon as it recovered from the narcosis. The wound healed by secondary re-union within five days. The cat was killed by chloroform six days after the date of the experiment.

The post-mortem revealed the presence of a jelly-like mass in the region of the operation. The stumps of the severed nerve were embedded in this mass. No pus was present.

I intended to examine the operated nerve by Marchi's method, but had the misfortune to lose it when washing it out after it had been hardened in the mixture of Müller's fluid and osmic acid.

#### EXAMINATION OF THE SPINAL CORD.

A series of transverse sections was made in such a manner that a continuous series of ten sections were mounted, then the following ten or twenty sections left out, then again ten mounted, etc. Staining with vesuvine in the same manner as in the case just described.

For better recognition of the outlines of the gray matter I left some of the thicker sections overstained. Be it said, however, that the paraffine sections are so ductile and change their shape so easily that the following statements concerning the gross anatomical changes must be taken with due reserve :

## PLATE IV.

(Exp. No. V.) Illustrating the alterations which follow the section of an intercostal nerve within six days.

FIG. 1. Photograph showing the dimension of the gray and white matter.

Magnifying power 13 lin. Vesuvine stain. (Overstained section.)

FIG. 2. Photograph showing the distribution of the diseased cells.

Magnifying power 47 lin. Vesuvine stain.

Deg. = District of diseased cells.

Back = Normal cells forming the group for the muscles of the back.

FIG. 3. Water color, showing various stages of the retrogressive cell changes.

Magnifying power 320 lin. Vesuvine stain.

# PLATE IV.

FIG. 1.

Operated  
side.

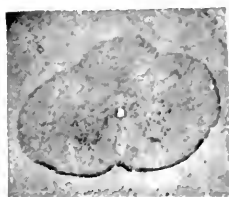


FIG. 2.

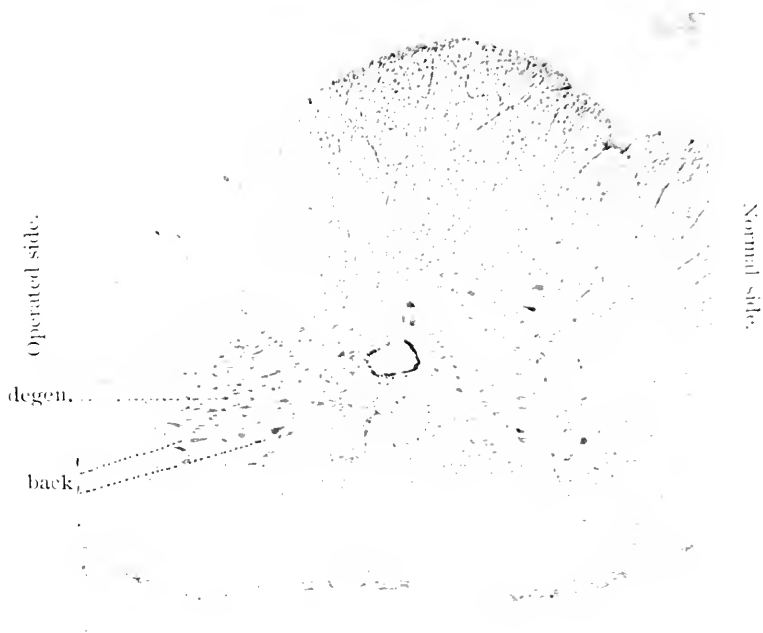


FIG. 3.



Fig. 1 reduced to  $\frac{1}{10}$  of size of original illustration.

Fig. 2 " " " " " "

Fig. 3 " " " " " "

The magnifying power is therefore

For Fig. 1 9 lin. (not 13 .)

For Fig. 2 37 lin. (not 47 .)





The left posterior column is enlarged in frontal direction. The same is the case with the left posterior horn. There does not seem to exist any differences in size between the antero-lateral columns and between the anterior horns. Regarding the anterior roots I dare not make any definite statement as they are too badly contrasted against the surroundings to permit judgment as to their calibre.

## I. GANGLIONIC CELLS.

### 1. *Cells of the anterior horns.*

There are distinct changes in the multipolar cells of the left anterior horn, but these changes are confined to a certain district which occupies the postero-external part of the anterior horn the cells situated at its anterior and internal border being mostly free from alterations, although also here diseased cells are occasionally met with. Fig. II. of plate IV. will serve to demonstrate the position and extent of the affected district.

The nature of the changes is the following :

The cells are considerably reduced in size. They have a shrunken dried-out appearance. This shrinkage affects all parts of the cell, including the nucleus and processes. To the processes of many cells it gives what Nissl rightly calls a corkscrew-like appearance. On account of their dark color (much darker than in normal cells) and of the hiatus which the shrinkage leaves around them they are better marked against the surrounding tissue than those of normal cells, and can be traced further peripherally.

The affected cells are much more darkly stained than the normal ones. In the beginning stages of the retrogressive process a granulated structure can still be recognized, the granula having an intensely dark tinge, but the granula (corpuscles) are less distinctly marked as the chromatophobe part of the substance of the cell-body is also quite deeply stained, while in normal cells it does not accept the stain. The cells resemble very much the type which Nissl describes as "chromatophile cells."

In many cells in which the shrinkage has become intense, a distinctly folded appearance, similar to that of a dried pear is noticed, in others it seems as if an asbestos-like splitting into rimes had taken place.

As was mentioned, the nucleus is also involved in the

shrinkage. It diminishes in size proportionately to the shrinkage of the cell-body, and assumes a dark color which in advanced stages corresponds to that of the cell-body so that its outlines become unrecognizable.

The nucleolus on account of the intensely dark color can still be frequently recognized when the outlines of the nucleus have become lost.

I feel satisfied that the principal characteristics of the observations described are a drying out and subsequent solidification of the cell. This is affirmed by the observation of cells in which one or several processes were found broken off and where the break had a serrated appearance, similar to the break of soft wood.

The said alterations could be seen as early as three days after the section of an intercostal nerve. At least quite a number of cells of the described character were found in the anterior horn of the cat upon which section of an intercostal nerve was performed, and which was killed three days after the experiment. As the series of sections was very incomplete in that case, it did not offer as good an opportunity to study the nature of the changes and the distribution of the diseased cells. In the cat operated upon in the same manner, but which was killed fourteen days after the experiment, the cell alterations, especially the shrinkage, were found much more advanced, a large number of cells had disappeared altogether, others were represented only by dark structureless chunks, others showed an exquisitely dark shrunken and folded appearance with thin, dark, cork-screw-like processes.

I have stated already that in the animal which was killed six days after the experiment (section of the intercostal nerve) the cells of the anterior and internal border of the left anterior horn were mostly found free from alterations. The district of affected cells occupies the central and external part of the left anterior horn. But as I mentioned before, in some sections single diseased cells were also observed at the anterior border of the anterior horn. In a few sections hardly one normal cell was found in the left anterior horn.

In the right anterior horn only few diseased cells were found; here they were not as distinctly grouped as on the other side; sometimes two or three shrunken cells were seen at the anterior border, then again one or two in the centre of the anterior horn, etc.

It must be mentioned that in the left anterior horn

the cells which underwent retrogressive changes belong on the average to a smaller type than those which were left free from changes. The diseased cells bear great resemblance to what Nissl calls chromophile cells. As the latter are met with in perfectly normal specimens in varying quantity and structure, N. warns not to mistake them for pathological formations. I must state, however, that in the case described the cells which presented the uncommon characteristics mentioned were so numerous and so typically arranged that to consider them as physiological formations is out of question.

Considering that in the last described experiment the nerve was severed peripherically from the dorsal branch which supplies the muscles of the back, and that the cells situated in the median and anterior part of the left anterior horn remained for the most part normal, it must be concluded that this normal group of cells supplies the muscles of the back. This agrees with the results of Kaiser, who states that the nucleus for the muscles of the back extends as a medial column through the whole length of the spinal cord. In position and extent this "medial group" (or medial column of cells) corresponds to the medial district isolated by the experiment mentioned.

Collins, whose investigations were based upon the examination of the spinal cord of an individual who in his childhood had been affected with poliomyelitis, has come to the same conclusion concerning the function of the medial cell group.

Spitzka's statement that the nearer the muscle is to the dorsal line of the animal, the nearer to the so called lateral cornu will its nucleus have to be sought for is, therefore, at variance with the findings of these experiments as it was with those of Collins. Furthermore, Spitzka's contention is denied by the fact that in experiment II, where both roots of a dorsal nerve had been severed all cell groups were equally affected, the medial group in no less degree than the others.

*2. Cell alterations of the character described were also found in the group of small cells at the base of the posterior horns.*

The area of diseased cells is situated more cephalad than the area of affected cells in the anterior horns. In many sections nearly every cell of the group at the base of the left posterior horn has undergone retrogressive changes. At the base of the right posterior horn less

diseased cells are met with, but still they are quite numerous.

The arrangement of the diseased cells in the course of the medial posterior root bundles makes it certain that the group is intimately connected with the posterior roots. It must also be assumed that part of the cells of this group are connected with the posterior roots of the same side, part with those of the opposite side.

3. *Cells of the medial zone (Waldeyer's Mittelzellen).*

On the left side many of the cells of this zone have also undergone the described retrogressive changes. On the right side only few altered cells are met with.

4. *Cells of Clarke's columns.*

The area of diseased cells is situated more cephalad than that of the altered cells of the anterior horns. Numerous cells have undergone the retrogressive changes reported. Besides this the absolute number of cells is diminished. In the group of the left side the alterations are more intense than in that of the right side.

5. *Cells of the lateral horn.*

In both lateral horns shrunken cells are found; considerably more in the left than in the right one.

## II. NEUROGLIA.

In some sections there seems to be a slight increase of the neuroglia elements in the left anterior horn. At any rate it is insignificant and in most of the sections cannot be stated. No signs of inflammation are noticed.

With regard to the localization of the cell alterations there exist some differences in the various experiments. In the spinal cord, which was treated after Marchi's method (section of anterior root of dorsal nerve) numerous degenerated cells were also found in the anterior horn *opposite* to the side of the lesion. In the animal experimented upon in the same manner, except that the spinal cord was treated after Nissl's method, only few degenerated cells were found in the anterior horn *opposite* to the side of the operation. The same was the case with the cat upon which section of an intercostal nerve had been performed.

This difference is probably due to the fact confirmed also by Nissl that the retrogressive changes do not set in simultaneously in all cells. The reason why more extensive changes of the cells of the right anterior horn

(opposite the side of the lesion) were found in the first case (Marchi specimen) is, evidently, that here the retrogressive changes had further advanced and involved a larger number of cells, the animal having been killed four weeks after the date of the experiment, while the other two animals were killed as early as six days after the date of the experiment.

Nissl properly calls attention to the fact that if purulent inflammations follow the operative lesion, many cells will be directly damaged by the inflammation, which would make it very difficult to decide what was the consequence of the inflammation and what the secondary effect of the lesion intended.

For this reason I excluded those cases from a special report in which I suspected that the retrogressive changes due to the interruption of the continuity of the nerve fibres were complicated with changes caused by inflammation.

Nissl confirms Weigert's statement that proliferation of the neuroglia towards the degenerating cardinal tissue (ganglionic cells and nerve fibres) is a normal reaction of the healthy tissue against the weakened one; but he adds that this proliferation varies very much in degree, being, for instance, quite insignificant for certain thalamus nuclei, while in other nuclei of the thalamus it is very intense. I, for my part, observed that the cell changes following the section of dorsal nerves or of their roots were accompanied by very insignificant, if any, proliferation of the neuroglia. It is possible, however, that proliferation of the neuroglia might have been detected at a later period, that is, if the animals had been killed say for instance two months after the experiment.

In summing up we find that the section of an intercostal nerve of a half-grown animal was followed by retrogressive changes both of cells intimately connected with the posterior roots of the severed nerve and of cells which give origin to its motor fibres. These changes could be made out within three days after the date of the injury, but they were more intense six days after the date of the experiment. They were marked by a gradual shrinkage of all parts of the cell accompanied by a modification of its structure and characterized by the increased affinity to certain aniline stains and evidently also by a solidification of its contents. It results from Nissl's observations that these alterations reach their

culmination within 18, 22–30 days after the interruption of the continuity of the nerve fibres. The lesion of the structure of the cell is much less deep, and the chance for recovery therefore much greater than in the homogeneous degeneration of the cell. Nissl, has indeed, observed that after the alterations have reached their culmination most of the cells partially recover by means of their lateral connections with other neurons. The chances for recovery of cells will, of course, be still better if the conditions for the regeneration of the peripheric nerve stump are favorable.

Howell and Huber who studied the degeneration and regeneration in peripheral nerve fibres after severance of the connections with their nerve centres, found that in no case a primary union between the fibres of the central and peripheral end took place. In all their experiments "degeneration of the peripheral end was complete throughout its entire length, (an observation confirmed also by Vanlair). The least time in which irritability began to return to parts peripheral to the cut was twenty-one days, and at this time regeneration was found to have progressed some distance beyond the wound."

It is very probable that while the peripheral ends of the severed peripheral nerve fibres undergo degeneration, retrogressive changes take place simultaneously in the nerve cells from which these fibres originate and that when the fibres, or better said, the axis cylinders of the central stump, begin to grow into the peripheric stump, the ganglionic cells also begin to recover.

In Exp. V (section of the intercostal nerve, animal killed six days after the injury) the conditions for regeneration of the nerve were certainly favorable, as the nerve was cut through without dislocation of the ends; still the physiological centres of the injured neurons underwent the retrogressive changes described.

The result of my investigations lead me to the following conclusions:

1. The severance of the continuity of a spinal nerve or of both its roots is always followed by retrogressive changes both in cells which are intimately connected with its posterior roots and in cells from which its motor fibres originate.

2. a. These changes may set in gradually and consist in a gradual shrinkage of all parts of the cell accompanied by a modification of its structure, characterized by in-

creased affinity to certain aniline stains and evidently also by a solidification of its contents.

b. Or the changes may set in acutely, being marked by swelling and homogeneous transformation of the cell contents; this transformation begins in a circumscribed part of the cell-body and then spreading itself towards the periphery, it involves the processes and nucleus.

This transformation may lead to liquefaction and final absorption of the cell, or may arrest itself at a certain stage.

In specimens treated after Marchi's method, the beginning of the homogeneous transformation is marked by swelling and distinct differentiation of all parts of the cell, while normal cells receive a shrunken rudimentary appearance by this method, so that their ganglionic character can hardly be recognized. In later stages of the degeneration the differentiation of the various parts becomes lost.

3. The character of the retrogressive changes is determined.

a. By the distance of the point of lesion from the cell.

b. By the character of the cell, or more probably by the manner of its connection with the severed fibres.

In "motor" cells severance of the motor fibre near origin from the cell (section of the anterior root) is followed by the acute changes, viz. by homogeneous transformation of the cell.

Lesion of the motor fibre at a considerable distance from the cell effects as a rule the gradual changes, that is a gradual shrinkage of the cell; but occasionally, as the case of amputation reported by Kahler and Pick proves, also here the changes in the "motor" cells may have the character of homogeneous degeneration.

In the cells of Clarke's columns, in the small cells situated at the base of the posterior horns, and in those of the lateral horns the changes always bore the character of the gradual shrinkage, no matter whether the nerve (larger distance), or the roots (small distance) was severed. Here the manner of the connection between the severed fibre and the cell which undergoes the change is less intimate than the connection between "motor" cell and motor fibre, as the latter is the principal part of the neuron of which the "motor" cell forms the centre.

4. The gradual shrinkage gives probably much more

chance for recovery of the cell, if the continuity of the severed nerve fibre can be restored, than the homogeneous degeneration.

5. The small cells situated at the base of the posterior horns along the course of the nerve bundles, which pass from the ventral part of the posterior columns towards the lateral columns, are intimately connected with the posterior roots. Part of these cells are connected with posterior root fibres of the same side, part with posterior root fibres of the opposite side. This connection probably consists in close contact of the terminal branches of the posterior root fibre with the network into which the axis cylinder of the altered cell dissolves itself; nothing positive can be stated in that regard, however, without examination by Golgi's or Ramon y Cajal's method. The latter makes no mention of said cells.

6. The cell group of Clarke's columns is in connection with fibres of the posterior roots both of the same and opposite side.

7. Of the cells of the medial zone (Waldeyer's "Mittelzellen"), and of the small cells of the lateral horn, I can only state, that they undergo a shrinkage after the severance of the peripheric nerve or its roots, but cannot decide with which of the roots they are connected.

8. The group of cells which supplies the muscles of the back with motor fibres, occupies the medial and anterior part of the anterior horn, which is in accordance with the views of Kaiser and Collins respecting the function of this group.

In conclusion I wish to recommend warmly Nissl's method of staining as a valuable addition to the methods in use for the investigation of the central nervous system. For the study of the finer structure of ganglionic cells it is much superior to the nigrosine, carmine and similar stains. This superiority is due to the fact stated by Nissl, that the hardening with the chromsalts damages the structure of the cells, for which reason fine cell changes will easily escape observation.

#### REMARKS CONCERNING ILLUSTRATIONS.

Part of the illustrations are photographs, part of them water colors. The first were made by myself, the latter by my sister under my supervision. The dimensions of the cells in the water colors were measured so as to represent the exact size corresponding to the power used.



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# INSANITY AND PHTHISIS, THEIR TRANSMUTATION, CONCURRENCE AND CO-EXISTENCE.

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IN 1892 Dr. Thos. J. Mays, of Philadelphia, read a paper before this society, in which he attempted to prove that pulmonary phthisis was essentially a nervous disease. While unable to agree with the conclusion reached by Dr. Mays, I was very much interested in the paper, because my own observation had furnished very similar data; but the conclusion I had drawn, and which I stated in the discussion of the paper, was different. My conclusion was that the concurrence and co-existence of phthisis and insanity indicated an imperfectly developed or unstable organism with a materially lessened power of resistance; and that this hereditary or acquired instability or vulnerability would manifest itself in the lungs of one individual, making him, under proper environment, liable to the development of phthisis; while in another, the instability being in the nervous system, the tendency would be to develop insanity.

In this paper I shall attempt to consider what constitutes this vulnerability, and why there should be such an intimate association between these two conditions, which are not necessarily concomitants, and which as a rule exist independently. However, the offspring of those individuals who are subjects of the tuberculous tendency are very likely to be insane. Indeed, so far as my observation goes, phthisis and insanity are equally potent factors in the production of brain instability.

During the past three years I have made as thorough a study of this subject as was possible with the opportunities afforded, modified, of course, in some cases by want of success in getting detailed information of the

life history and hereditary tendencies of the individuals studied. In the beginning I found it useless to attempt to go beyond the time of my personal knowledge of the cases in the hospital, because before that time no effort had been made to get an accurate life history of the individual patient, and there was no record of any physical diseases from which he might have suffered.

During the period since August 1892, out of 695 patients admitted, 70 had a history of phthisis in the family. Thirty-one of these were men, and thirty-nine were women. During this time fifteen men and four women have died from phthisis, and of this number twelve men and one woman were residents of the hospital for a longer period than two years.

The following tables will give the distribution of these cases with regard to hereditary manifestation. In the first tables I have included the admissions in which there was a history of phthisis in the family, and I have described those cases as insanity due to transmutation, in which there is a history of direct or collateral heredity of phthisis, but not of insanity (however in three of the cases a combined heredity existed), while those cases in which phthisis existed in the brothers or sisters of the patient are described as concurrent.

In these tables two general subdivisions have been made. The patients have been classified as primarily degenerate when the insanity has manifested itself before 35 years of age, and progressed steadily toward terminal dementia; as unstable when the degree of defect has not been so great, and the perversion has been temporary, or has made its appearance after middle life, and consecutive to somatic degeneration in some form.

The apparent discrepancy in the figures for degenerate women is due to the fact that some of them had both a direct and collateral heredity.

#### TRANSMUTATION AND CONCURRENCE.

		Direct.	Collateral.	Paternal.	Maternal.	Concurrent.
Men,	31					
Degenerate,		4	9	6	7	6
Unstable,		8	1	2	7	8
Women,	39					
Degenerate,		14	7	6	12	4
Unstable,		6	6	4	8	5

The preponderance of degeneration among those

having an heredity of phthisis is significant, as indicative of the influence of phthisis in one generation, in determining a defective nervous system in the next, and these tables also indicate that the more direct the heredity the greater is the probability of transmutation. The apparent discrepancy between the sexes as to the number in whom the heredity was direct, I cannot explain, but believe it to be accidental. The relatively large number in whom insanity and phthisis was concurrent among brothers and sisters is also of great importance, as showing the intimate association between these forms of degeneration.

The following tables show the co-existence of insanity and phthisis both among the living and the dead, and indicate the time of the tubercular development.

## CO-EXISTENCE AMONG THE LIVING.

		At time of Admission.	During First Year.	In Dementia.
Men,	5			
Degenerate,		2	1	1
Unstable,		1		
Women,	7			
Degenerate,			2	3
Unstable,				2

## CO-EXISTENCE AMONG THE DEAD.

		At time of Admission.	During First Year.	In Dementia.
Men,	15			
Degenerate,		1	2	7
Unstable				5
Women,	4			
Degenerate,		2		1
Unstable,			1	

With regard to the class of cases figured in these tables it might be well to note here that, like all old-style hospitals, we have the *materies morbi* for tubercular infection always with us, and, as noted, the majority of our cases develop phthisis after coming to the hospital.

The following tables show the form of heredity in those who have died of phthisis, and among those living who are suffering from the disease.

## FORM OF HEREDITY IN THOSE DYING OF PHTHISIS.

MEN.				WOMEN.			
Insane Heredity.	Heredit. of Phthisis.	Both.	Unknown.	Insane Heredity.	Heredit. of Phthisis.	Both.	Unknown.
4			11	1			2.

## FORM OF HEREDITY IN THOSE LIVING AND SUFFERING FROM PHTHISIS.

MEN				WOMEN.			
Insane Heredity.	Heredity of Phthisis.	Both.	Unknown.	Insane Heredity.	Heredity of Phthisis.	Both.	Unknown.
1	3		2	3	1	1	4

In those cases in which there is no history of the form of heredity, the presumption, reasoning from analogy, is in favor of an heredity of insanity. It is especially worthy of note in this connection, that of those dying of phthisis not one had an heredity of that disease, while among those living three men and one woman had an heredity of phthisis. Going back to the first tables we find further proof of the transmutation of these two degenerate conditions in the fact that of the seventy cases having an heredity of phthisis only three men and two women have developed the disease.

The three following case records furnish evidence of the co-existence and transmutation of these two diseases, and illustrate the types of defective organization upon which they are engrafted. The record in the cases of the two children is given in detail because they furnish the data for the argument I wish to make as to the nature of the changes which bring these two forms of somatic defect into such intimate association.

The first case illustrates the co-existence of phthisis and insanity in a defective individual in whom the symptoms of active mental perversion were probably antedated by the tuberculous process, although no definite history of the onset could be obtained.

S. O., admitted July 26th, 1892. No history of insanity or phthisis admitted, but the father used alcohol to excess when a young man. The other children, one older and three younger, are healthy, and possess ordinary intelligence. This child was of good disposition before puberty, but since that time has been morose and more or less irritable. She was born in Norway, and came to this country, only a few weeks before admission, with her family. Soon after her arrival her irritability increased, and she became violent. She was nineteen years old when admitted, had menstruated once at the age of fifteen, but not since, and the menstrual molimen was afterward represented by epileptoid convulsions, which continued up to the time of her admission to the hospital. She was quiet and stupid, took no care of herself and had to be fed. About a week

after admission she began to cough and had some elevation of temperature. The cough and expectoration increased, the temperature rose steadily, and she began to suffer from diarrhœa and profuse sweating. The temperature reached 105° on the sixth day of her illness, and kept at 103° until she died. The respiration ranged between 34 and 60, and the pulse between 96 and 142, the diarrhœa continued throughout, and the sweating was at all times profuse. No marked symptoms of pulmonary involvement were present at any time during her illness. Her stupidity increased, and finally deepened into coma and death. Died July 25th, 1892.

The postmortem, as was to be expected, disclosed general tubercular involvement. The dura was not thickened, but the subdural space was distended with fluid, especially over the frontal lobes anteriorly. The vessels in the pia were very much injected over the motor area, in the fissure of Sylvius and in the sulci, where the injection was greatest. Numerous small nodular masses were found in the velum interpositum and along the choroid plexuses. The pleura on the left side was adherent throughout the entire surface. In the apex of the left lung were small cavities filled with pus. There were no adhesions on the right side, but at the base of the right lung there was marked hypostatic congestion, and in the middle lobe were several small cavities filled with pus and surrounded by softened tissue. There was an increase of fibroid tissue in both lungs. The mesenteric glands were enlarged, especially in the region of the cæcum. The lower portion of the ileum and cæcum were very much injected, and this portion of the intestine was studded over with enlarged solitary glands, while Peyer's patches were thickened, and in the neighborhood of the cæcum ulcerated. The liver, spleen and kidneys were not involved.

During the past two years I have had an opportunity to study two children of insane parents, who died of tubercular meningitis, and these cases have been of especial interest to me, because they illustrate the two kinds of degeneration that I wish to discuss.

I. C. M. Female. Was born in this hospital May 20th, 1892. Both parents were insane, and the mother had been a dement for some years. The history of the father shows him to have been mentally defective. He had been twice in this hospital, at intervals of a year, suffering from maniacal excitement, which subsided

rapidly. He, however, remained unstable for some months afterward in both attacks. He was 23 years old at the time of his first admission, 25 at the birth of the child, and had suffered from two attacks of mental excitement during the preceding five years, beside being incorrigible and very dissipated. This young man was born in Canada, and no history of his family could be obtained; but it was ascertained that his own mental instability began to manifest itself soon after puberty, thus determining its character. Physically he was very robust, and without the slightest indication of organic visceral disease.

The mother, 37 years old at the time of the child's birth, was very robust physically, but had been stupid and demented for a long time. She had always been well physically, but never very bright, and there was a history of collateral heredity, one of the brothers of her mother having been insane.

There was no deformity or apparent asymmetry present at the time of birth in the child, and the mother nursed it during the first eight months of its life. During this time the child was apparently robust, although it was noticed that there was a tendency to disproportionate development of the head, especially in the parietal region. Also that the two sides were not symmetrical, and the vertex was flat. About this time it was also noted that the little girl had a singularly transparent skin, with the superficial veins large and distinctly defined, especially at the temples over the head and on the chest and abdomen.

From its eighth month on, as the mother had ceased to care for it, the little girl was nourished artificially, and although not so robust as formerly was still apparently healthy. The child was singularly beautiful, with large expressive blue eyes, clear transparent skin, rosy cheeks and light hair; its facial expression being what mothers are apt to call "angelic."

The first symptoms noticed in her last illness were an apparent weakness of the legs and an indisposition to walk (she had always been very active). This occurred about the last week in July 1893. During the latter part of the month and the first week in August she would from time to time roll her head in the pillow, usually while lying down and after waking from sleep. This disturbance was at the time attributed to the fact that her molar teeth were at the point of eruption.



Soon after the first symptoms appeared a diarrhœa set in, the stools being fairly consistent, and she also vomitted occasionally at this time. The symptoms did not respond to treatment, and the diarrhœa and vomiting in varying degrees of severity were present during the whole of her illness. Periods of constipation were intercurrent, and for a time the child had colicky pains. On the 6th of August she suddenly became quite stupid, lying quietly with eyes closed, and only looking up after loud calling or persistent effort to attract her attention. This stupor, almost continuous for several days, was later attended by intervals of apparent return of intelligence, when she seemed to recognize those around her. Later there was marked motor disturbance, with rolling of the head and convulsive movements of the limbs; at first limited to the hands and face, but gradually becoming general, when the head and eyes were turned to the left. She also had screaming spells, and moaned a great deal during the last few days. The temperature varied between  $101^{\circ}$  and  $103^{\circ}$  F. Pulse between 110 and 160. The respiration was very much accelerated, and examination of the lungs at various times during the last week showed varying rales and irregularity of respiratory murmur. Areas of harshness and impaired resonance were also present. The child was practically unconscious during the last week of its life, with frequent convulsions which occurred as a rule at night. The skin became waxy, and the blood vessels stood out distinctly outlined under the skin. She died August 13th, at 7 A. M.

Autopsy six hours after death. Body pale, waxy in appearance, and very much emaciated. Rigor mortis absent. Cadaveric lividity present, and the fingers and toes cyanosed under the nails. The eyes were sunken, pupils moderately contracted. The anterior fontanelle was present, with the scalp sunken in over it, but the posterior fontanelle had disappeared.

The circumference of the head was 47 cm., occipitonasion, 29.9 cm., occipito-mental, 51 cm. Bi mastoid diameter, 30.5 cm., bi-parietal, 18.4 cm., right oblique, from frontal eminence to parietal boss, 24.1 cm., left, 24.8 cm. Length of body, 76.8 cm. There was no asymmetry in body or limbs.

The scalp externally was smooth and free from scars and depressions, but less than the normal amount of hair was present. The scalp was free, smooth on its in-

ternal surface, and contained a normal amount of subcutaneous fat.

The skull was smooth, externally and internally the sutures all present, grooves for sinuses and vessels normal, bone thin and elastic, diploe only present in the temporal region. The dura was adherent over the vertex and bulging with fluid. Its external surface was smooth laterally and anteriorly, but rough and thickened over the vertex. The internal surface was smooth but adherent over the right occipital region to the pia-arachnoid, where it was granular in appearance, presenting when separated a surface studded with small white tubercular bodies. The longitudinal sinus was empty, but the lateral sinuses were filled with blood. The quantity of cerebro spinal fluid was 6 oz.

The pia-arachnoid was translucent in general appearance, thickened, and the thickening most marked in the Sylvian fissures. The membrane was very much distended with fluid, and was adherent to the dura at the base anteriorly, where it was very thick and white, being studded with miliary tubercles. The membrane was not adherent to the cortex, but its vessels were enormously distended.

The brain weighed 36 oz. and was softened. All over the convexity there were patches of opacity along the vessels, most marked anteriorly and near the median line. The fissures of the median surface were well developed, likewise both Rolandic fissures. The convolutions were simple and the sulci shallow. The simplicity was most apparent in the anterior lobes, while the greatest complexity existed in the parieto-occipital region. The frontal lobes were adherent by their proximal surfaces at the base.

The lateral ventricles were distended, and their ependyma thickened and opaque, but no tubercles were apparent on the surface. The velum interpositum and choroid plexuses were thickened and studded with miliary tubercles. There was a large thick mass extending along the hippocampal lobe on the right side, made up of a cord-like membrane, accompanying the choroid plexus to the base of the brain; apparently an extension inward of the thickened pia at the base. This thickening existed, but only to a slight degree, on the left side.

The corpora quadrigemina were small in proportion to the other nuclei, and the ependyma of the third and fourth ventricles was thickened, opaque, but not rough-

ened. The brain mass was softened and the puncta were well marked. The cortex was thin, pale and translucent, being least well developed in the occipital, thicker in the parietal lobes, and thinner anteriorly. The tracts in the pons and medulla were beautifully defined externally. The pons, medulla, and cerebellum did not share in the softening of the brain mass.

The anterior and posterior mediastinum with the pleural cavities were not diseased. The pericardial cavity contained about 1 oz. of fluid, and its surface was smooth.

The lower lobe of the left lung was congested, and miliary tubercles were present in it and at the apex. The posterior portion of the upper lobe of the right lung was congested, and tubercles were scattered irregularly throughout its substance.

The heart weighed  $1\frac{1}{2}$  oz. The left ventricle was thickened, the foramen ovale incompletely closed, and a fat clot filled the right auricle extending into the ventricle.

The omentum was drawn up and shrunken, the mesenteric glands enlarged. The spleen weighed  $\frac{1}{2}$  oz., its capsule was adherent, and its substance was studded throughout with miliary tubercles. The liver weighed  $12\frac{1}{2}$  oz., and showed no sign of disease. The left kidney weighed 1 oz., its capsule free, the pyramids well marked and the cortex normal. The right kidney weighed  $\frac{3}{4}$  oz., capsule free, pyramids well marked, cortex normal. Both kidneys were lobulated.

I. H.—Born February 9, 1894. Died October 26, 1894. At birth was apparently strong and vigorous; was well nourished and weighed  $8\frac{1}{2}$  pounds. Was nursed by mother until the early part of July when he was weaned on account of the mother's failing health, and the consequent poor quality of her milk. When taken from the breast he had considerable elevation of temperature, and for several days thereafter, stools were abnormally frequent, and were green in color. Some premonitory signs of meningeal congestion were present. Artificial feeding was substituted. Upon this he steadily gained and when seven months old, the middle incisors of the lower jaw were erupted without any nervous disturbance. Shortly afterwards he became more fretful, crying a good deal. He took his nourishment in normal amount, however, and temperature remained normal; stools were also normal in character. This condition continued

until October 1st, when he became markedly quiet and stupid. Much of the time he lay sleeping, occasionally waking with two or three short, low outcries, and then falling asleep again. His temperature rose to  $100^{\circ}$  or  $100.5^{\circ}$ , and did not vary much from that until October 18th, when it reached  $101^{\circ}$ . His pulse was somewhat rapid, ranging from 116 to 130, quick in character. On October 18th he had two convulsions both most marked on the left side of the body. The head was turned towards the left side. An alternating strabismus present to slight degree at all times since birth, became exaggerated during convulsive seizures and subsequent stupor. Coarse tremors and irregular muscular contractions were present in the intervals of the convulsions, these movements more pronounced on the left side. From this time onward he took nourishment poorly. Bowels still moved in response to an emulsion of Oil. Ricini.

The day following he had two convulsions in which the twitching was most marked in left arm, and only slight in left leg. Body assumed a position of opisthotonos, and retained it until relaxed in mustard bath. At intervals during the day had tonic spasms, but the clonic movements were absent. On the 20th inst. respiration was irregular, pseudo-Cheyne-Stokes in character; there were four groups of five or six respiratory acts, to the minute, these groups being separated by periods of apnoea. Head was bathed in copious perspiration. Pupils were decidedly unequal, the left being larger. The left eye was in a state of internal squint. Conjunctivæ were not responsive to direct stimulation. Nourishment was taken in very small amount.

October 21st. Marked evidence of effusion, from the distention of the calvarium, bulging at anterior fontanelle. Measurements at this date are as follows:—

Biparietal Diameter . . . . .	13 cm.
Nasion to Inion . . . . .	28.5 cm.
Biauricular Line . . . . .	29 cm.
Left Temp. to R. Par. Boss . . . . .	20.3 cm.
Rt. Temp. to L. Par. Boss . . . . .	18 cm.
Circumference through Inion and Nasion . . . . .	44.2 cm.
Circumference through Obelion and Ment. . . . .	49 cm.

At this time both pupils were responsive to light.

From this date onward the emaciation progressed, and the amount of food taken diminished. On the 23d inst. he had a general convulsion, and marked opisthotonos on the 26th inst. His body then became quite cyanotic in appearance, eyes were sunken and extremi-

ties cold. The temperature continued to rise higher and was but slightly influenced by the application of cold during the last three days. He died October 26th.

Autopsy:—One hour after death. Body warm and fairly well nourished. The cranial cavity is very much distended and the head gives the sensation of greatly increased weight when held in the hand. A tendency to contracture in the flexor muscles of the fingers persists so that after the fingers are straightened out, they immediately return to a position of contraction with the thumbs drawn into the palm of the hands. The feet were everted, the left more so. There is a tendency toward mottling of the skin, beginning on the inside of the arm and in axilla. (Before the examination was completed this mottling was general over the whole body.) There was no asymmetry of the body except a slight bulging of the chest wall on the right side anteriorly.

The pupils were unequal, the right measuring 4 and the left 5 mm. The eyes were sunken, but this is partly apparent on account of the protrusion of the frontal bone. The skull is Brachycephalic and the facial line almost perpendicular. The coronal suture is united but the sagittal and lamboidal are loose, there being a separation of 4 mm. between the parietal bones on the median surface and an overlapping of the occipital by the parietal of 2 cm. Posterior fontanelle absent. Anterior present measuring 4 cm in lateral and 2 mm. in longitudinal diameter. The body measured 69 cm. Circumference of chest 37.5 cm.

Measurement:—Occipito nasion, over convexity 28.2 cm. Circumference 48.2 cm. Bimastoid over vertex 26.5 cm. Verto mental 48.2 cm. From right frontal eminence to parietal boss 17 cm. From left to right 15.2 cm.

The dura was adherent to the bone along the line of the sutures, and in the parietal bones the adhesions extended over most of the surface, also over the frontal parietal and occipital bones on the left side, while on the right side the membrane was free except at the margins of the fissures. The dura in the fossæ was very thick and adherent, especially in the temporal portion of the middle fossa. The dura was generally thickened and its vessels filled with blood. There were 4 oz. cerebrospinal fluid free in the fossæ and the membranes were all matted together at the base, posteriorly and infer-

iorly, making it necessary to dissect out with the finger the pons, medulla, and cerebellum.

At the base there was a complete effacement of the superficial anatomical subdivisions, and even the blood vessels were included in the network of new tissue formation in the pia-arachnoid. This extended around the anterior border of the cerebellum and also into the Sylvian fissures, in the left one of which a membranous meshwork was formed 3 mm. in thickness, and which could not be dissected out without tearing the cortex in the region of the operculum and insula. The cranial nerves, where not involved in the new growth, were softened. All of the surfaces described were studded with miliary tubercles varying in size, from 1 to 3 mm., while the pia-arachnoid covering the cortical portions at the base was studded with finely granular tubercles. This same condition existed all over the convexity. Along the median fissure, especially in the frontal and parietal regions, the arachnoid was thickly studded with tubercles, and they extended into the fissure down to the corpus callosum, on the surface of which, and springing from the matted pia arachnoid, were three tubercles, varying from 1 to 3 mm. in diameter, also numerous granular ones. Over the convexity in the parietal region, and especially on the left side, were numerous creamy patches, but, except in their immediate vicinity, the pia-arachnoid was not thickened, and was opaque only along the vessels.

The principal fissures were illy defined and shallow, and the convolutions were simple, flattened and disposed to be confluent. The lateral and third ventricles were much enlarged and distended. The left had the greatest capacity, and both approximated the size of the ventricles in an adult brain. The ependyma in all of the ventricles presented a granular surface, and the blood vessels were deeply congested. The floor of the fourth ventricle was covered with a filmy meshwork of newly-formed membrane, and this extended along the nerves, having their origin back of the corpora quadrigemina. The velum interpositum and choroid plexuses were adherent, thickened and studded over with granular tubercles, while in the ependyma of the left lateral ventricle over the centre of the caudate nucleus was a tubercle 4 mm. in diameter. The approximate surfaces of the cerebrum, pons, medulla and cerebellum, were matted together, and the membranous network which

united them was filled with granular tubercles. In the centre of the surface of the vermiform lobe was one tubercle which had undergone caseous degeneration, 10 mm. in diameter, and on the lateral lobes on each side were other tubercles, varying from 5 to 8 mm. in diameter.

The brain mass was soft, its cortex varying from 2 to 3 mm. in thickness. The nuclei were as large as are usually found in the ordinary adult brain, and the whole mass weighed 29 oz.

The heart weighed 1 oz. A small amount of fluid in pericardium. No tubercles or inflammatory products, but the coronary vessels were kinked and relaxed. A little fluid blood in left ventricle and a black clot filling the right auriculo-ventricular cavities. The aortic and pulmonary semilunar valves were competent and normal, and the tricuspid and mitral valves appeared normal. Aorta not abnormal in appearance. Heart muscle seemed normal, and so the endocardium.

Right lung weighed 1 $\frac{3}{4}$  oz. Strongly adherent at apex. Caseous mass in upper lobe posteriorly 20 mm. in diameter, not broken down. Miliary tubercles scattered through entire lung, especially posteriorly. Left lung weighed 1 $\frac{1}{4}$  oz. Miliary tubercles scattered throughout upper and middle portions. Lower lobe most involved.

The spleen weighed  $\frac{1}{2}$  oz., and was full of miliary tubercles. Adjacent portion of pancreas also full of them.

Liver weighed 7 oz. Granular tubercles scattered through its substance. Gall bladder distended with bile. Ducts patulous. Supra renal capsules studded with tubercles. Normal amount of fat about the kidneys, and considerable fat in the walls of the abdomen. Right kidney weighed  $\frac{3}{4}$  oz. There were tubercles scattered over the surface. Capsule free. Left kidney weight 1 oz., presented the same physical characteristics. Bladder distended; did not appear abnormal.

A few granular tubercles in peritoneum, where in contact with liver. The stomach appeared normal. Mesenteric glands enlarged, measuring from 5 to 10 mm. in diameter, and tubercular. The small intestines, especially the jejunum, and also the colon, were studded with tubercles, which in places almost produced strictures of the intestine, forming circles of tubercles around it, and seemed to be ready for ulceration. The diaphragm contained numerous tubercles.

In the case of the first child both parents were typical defectives, and the mother is now a stupid dement with arterio sclerosis developing. The child represented a typical transmutation. Large brain, with excessive development in the parieto-occipital region, mental and physical precocity, and when her illness began, symptoms of cortical irritation from the first with marked and excessive explosions of motor-energy. Again, post mortem, is found destruction of functional tissue rather than excess of tubercular deposit in connective tissue.

In the second child the history of the parents is not definite so far as known. The child was illegitimate, and the woman had given birth to three others, two of whom had died. One is living and robust.

The mother has developed tuberculosis by infection since the birth of this last child.

The life history of this child is exactly the opposite of the first. Stupid and quiet from birth, dull and uninteresting, skin thick and coarse, and without color. Signs of degeneration appeared early in twitching and disturbed sleep. The rapid progress of the tubercular disease after its onset, also the enormous effusion, which, however, disappeared in part before death. Post mortem, instead of ulceration and destruction of mucous surfaces, an almost universal infiltration of connective tissue with tubercle, also marked connective tissue increase.

Degeneration naturally takes place in connective tissue or epithelial, and is necessarily primarily existent in one or the other. That is, if degeneration begins in an epithelial structure the connective tissue stroma will not undergo degeneration until by contiguity it has become affected (first proliferating and then degenerating because its excessive growth has reduced its potentiality for reconstitution). Also when degeneration begins in a connective tissue, the epithelial tissue is not affected until either mechanically by pressure or indirectly by occlusion of blood vessels and lymph spaces, the nutrition of the epithelial tissue is cut off.

For my purpose in argument I will call degeneration of the epithelial cell—functional destruction, and that of the connective tissue stroma—structural degeneration.

They are both characterized, first by a rapid proliferation of imperfectly formed cells, and finally by des-



truction of tissue, the nature and product of the destructive process being dependent upon its immediately exciting cause.

Among the insane epithelial degeneration is very uncommon as a primary form, although it can occur in their descendents, as seen in the first child. Among degenerates we find that out of all those subject to infection only a certain number become the victims of tuberculosis; also, it is a well known fact that among victims of phthisis only a small number become insane. Out of 695 cases admitted to this hospital during  $2\frac{1}{2}$  years only two men and three women were suffering from phthisis when admitted.

The two children, whose cases are reported, illustrate not only the transmutation from one form of defect to the other, but also the two forms of degeneration, the girl representing the epithelial and the boy the connective tissue type.

In the development of the ovum primarily there is no morphological difference between the cells of the two germinal layers, nor is there anything distinctive at the time of the formation of the mesoblast, and yet from both the inner and outer layers everything that is functional in the organism is developed, while the mesoblast furnishes the framework and connective material for all the organs of the body. The morphological characteristic of all functional tissues of the body is the epithelial cell as the functioning apparatus, and the connective tissue cell as the framework in which it rests. During the physiological activity of a functional tissue the epithelial cell disintegrates being reduced to a simpler form or undergoing retrograde chemical changes. In the connective tissue, however, the process is one of reconstitution *without change of form*, as illustrated in the changes which take place in unicellular organisms. The recent work of Weismann gives an elaborate description of these processes, but from another standpoint.

The epithelial cell is a finished product of organization, and develops into nothing else, although it assumes different forms according to its locality. The connective tissue cell, however, not only undergoes changes in form, but also in structure, so that in its highest stage of development it ceases to have any definite structure or capacity to reproduce itself. Strictly speaking, however, this change from cell to fibril would be one of degeneration, although not of disintegration,

such as takes place in the epithelial cell. Now as to the tissues formed by these two types of cell, the epithelial, being the least organized, is the most unstable, being easily broken up with the liberation of considerable energy and the formation of a lower type of cell still more simple and unstable, as illustrated in the cells of the different layers of the cortex. The connective tissue on the contrary is highly organized, stable, not easily affected by incident forces, and having no function in the life processes of the organism; furthermore, the only change it undergoes when deprived of nutrition is liquifaction, as seen in the contents of cysts, especially those occurring in fibroid growth.

It has been my observation, especially among the insane, that disease processes, which are constitutional or diathetic, attack primarily one or other form of tissue with the result of progressive degeneration and disintegration if the tissue be epithelial. However, if the connective tissue is the seat of a diseased process (strictly speaking subject to excessive irritation or withdrawal of nutrition), it either increases rapidly in amount, and remains permanently increased, or undergoes liquefaction.

I have also noticed that among the insane, according to the degree of defect in organization, that there was excessive development of connective tissue with a tendency to rapid proliferation from slight sources of irritation or else extreme response to irritation in epithelial tissue, resulting in rapid disintegration. The most common form of degeneration among the insane is the connective tissue type or the premature and excessive manifestation of the changes, which ordinarily occur in senility. The disproportion between the connective and epithelial tissue constitutes the defect, the amount determines the degree of defect, and its manifestation is in degeneration, as idiocy, imbecility, the higher form, paranoia, or that form represented in those individuals whom Lombroso calls *mattoids*. In all of these there is a tendency toward premature senility with the development of atheroma, increase of fibroid tissue in all organs and the wasting of epithelial tissue by pressure from encroachment. Of these types the one in which phthisis is most likely to be developed is the lowest grade, the idiot and imbecile, and this is indicated by the statistics of institutions for the care and training of the feeble-minded, which show a large

percentage of deaths from phthisis. This is also the case among the insane, and even among those who have remained free from tubercular infection we find, post mortem, marked degenerative change in the pleural cavities and parenchyma of the lung. Out of 111 deaths in this hospital during the biennial period fifteen were from phthisis, thirteen men and two women.

Now what is the biological connection between these two forms of defective development? I have made the statement at different times that I did not believe that the parents should have exhibited a form of defect in order to consider its manifestation in the child as the result of heredity: and that this was especially so in insanity, where the unstable or defective nervous organization of the child may have had for his parental antecedent gout, rheumatism, syphilis, phthisis, or even some profound interference with nutrition during gestation, without the previous existence of any diathetic condition in either parent. I need only add that my further experience confirms this belief.

Next to the functional nerve cell there is no other form of epithelial cell so unstable as that of the functional tissue of the respiratory and digestive apparatus, and then these three are not only more directly and intimately associated, but exist together, even before a circulatory apparatus is developed. In fact the primitive manifestations of life are irritability and respiration.

Although none of the hypotheses concerning heredity and development are fully accepted by all, still they are agreed as to the influence of nutrition and the tendency of parts of the organism to be arrested or limited in their development. As the constituents of the ovum break up into the cell layers, the tendency is to develop in relative proportion. The epiblast and hypoblast appearing sometime before the mesoblast. From the epiblast develops the skin, nervous system, etc. Most authorities also assign the epiblastic layer as the source of the mesoblast, from which in turn develops the connective framework, the circulatory system of the organism, muscles, etc. Now from what we know of heredity, although the direction of development is determined, its completeness is dependent upon nutrition; so that although all parts will exist, if nutrition is imperfect, the most vigorous will develop first and at the expense of the least vigorous; with the tendency for the less vigor-

ous to persist in an imperfect form. This is illustrated in plant life, when if the seed is sown too thickly there is an enormous development of stalk, with only stunted and imperfect grain and if added to this there is not sufficient moisture, the plant will develop prematurely and die without the production of seed.

Now if through any of the numerous causes of impaired nutrition and lowered vitality in one generation, the development of the ovum is interfered with, we find the result in the next generation in the form of the defective individuals I have described; manifesting in various degrees the tendency toward premature senility, with either sterility or the excessive procreation of defective offspring as in plant life. The nervous system and vegetative organs being most highly organized will suffer most, and when the defect is great we will have a preponderance of imperfectly developed connective tissue, while in higher forms there is an excess of epithelial or functional tissue which being imperfectly developed is more unstable and disintegrates more easily. These are the changes which to my mind explain the intimate association of insanity and phthisis and their co-existence; while according to the laws of heredity, the preponderance of imperfectly developed connective tissue in one generation implies the excessive development of functional tissue in the next, thus accounting for the transmutation of disease tendency. These changes are abundantly illustrated in the tuberculous and defective children of neurotic, gouty, or syphilitic parents.

Of course, these processes may be modified in many ways, and I do not include those cases of defective or irregular development which are due to causes operating upon the child during pregnancy, accidents during labor, or the many conditions operating during the two first years of life to produce arrest of development.

The intimate association between phthisis and insanity, however, is certainly significant, and the observations I have made could undoubtedly be paralleled in any institution for the insane. The weak point in the paper is my inability to present a similar series of observations of the nature of the changes found in those dying of phthisis who are not insane, and I have not been able to find anything bearing upon the subject, from this standpoint, with which to compare my observations. The points especially requiring explanation are the infrequency of insanity among victims of phthisis and the

nature of the hereditary conditions in these cases. It is certainly a fact that many cases of phthisis, who do not become insane show some of the stigmata of degeneration, but I am debarred from studying this part of the subject in detail. I hope, however, that the subject as I have presented it will interest some of you who may have this opportunity, for I am growing more and more confident that the pathology of insanity will only become clear when we fully understand the nature and causes of defective development. It also seems to me highly probable that aside from the asymmetry and undue or imperfect development of the external parts of the body, we have as the principal characteristic of somatic degeneration excessive growth of connective tissue; while in the higher form of instability or irregular development, the most marked characteristic is excessive development of highly unstable functional tissue easily disintegrated and of limited potentiality, with connective tissue increase as a secondary manifestation.

*(The Proceedings of the Twenty-first Annual Meeting of the American Neurological Association will be continued in the November Number.)*

## Asylum Notes.

*Investigation in New York.*—We are in receipt of a pamphlet published by a sub-committee of the Senate Committee of Finance, which investigated both the Lunacy Commission and its relation to the State Hospital service. The report commends quite unqualifiedly the work of the Lunacy Commission, especially in reducing the expenses of the State institutions by the amount of \$300,000 in the year 1893. It also claims that this has been done without any depreciation in quality of goods or service. It publishes the compensation which the board receives, the medical member receiving \$5,000 with a \$1,200 allowance for traveling expenses, the legal member \$3,000 and \$1,200 for traveling expenses, and the lay member \$10 per day of actual service and \$1,200 for traveling expenses. It speaks of the work with unreserved commendation.

It then turns to the State local hospital service. It calls attention to the alleged former irresponsible and unchecked form of the service,—to the prevalent location of each hospital regardless of whether or not it was nearest the largest number of patients; to the immense sum of \$13,000,000 spent since 1870 for an increase in the number of beds; to the claim that each board seeks regardless of the others to get all it can for its own locality; to the fact of inequality of the salaries of officers and employees who do the same service; and calls attention to the allowances of the superintendent over and above their salaries, of a living which is not limited in cost or quality by any specified bounds. It also notes the rumors of favoritism in wages, in allowances and in purchasing of supplies, and recommends the equalizing of salaries for equal services and the giving to the officers a distinct salary instead of the variable perquisites of lodging, fuel, horses, servants, etc.

In comment upon the above we do not wish to criticize local conditions, of which we may not be sufficiently well informed; nor do we wish to try and straighten out this (as we are persuaded) somewhat one-sided report.

Only one thing which is made very prominent in this report would we try to modify. The tables which show the high salaries, for example, of the superintendent

of from eight to ten thousand dollars per year, are unjust. To show this, we might in like manner take the case of one of these very legislators who make up this report, who we will say is getting a nominal \$500 or \$1,000 per year. If we would add to his salary two or four thousand dollars a year, as his share of the rental and keeping up of the Albany capitol building and its expensive plant, and then criticise his salary as too extravagant, he would protest at once and rightly. The truth is, the State is apt to honor itself by costly buildings which should not be added to the man's salary who is chosen to serve in them. This is over and above the inconsistency of estimating a ten per cent. rental, and to this adding repairs and expenses which such per cent. should cover. These estimates will stand a very generous scaling down at the very least.

It only just to add that the nominal yearly salary of a superintendent in the United States is probably only about \$2,500, not equal, we think, even with the living usually added, to the income of many of the ordinary country practitioners about them. Although the State may expend more money than this, yet the ordinary living of a superintendent is probably worth only from \$1,000 to \$1,500 per year, and that of the assistants from \$400 to \$800 per year.

*Trained nurses to travel with patients.*—To have patients who are insane brought as patients by persons who are with the uniform and training of nurses, instead of being brought as prisoners and by officers of the law, would seem to be very rationally and easily defended. Yankton, South Dakota, has tried and maintained the value of this in its last report, and also its great economy. The New York Lunacy Commission has ordered trained attendants to be sent after patients in that State, due notice being given and seemingly as far as we can learn with good results. As a matter of incidental value it may be said that there is a considerable saving to the State as a whole, though the pay comes through different channels and thereby takes away a very profitable industry from the sheriff and adds a slight advance to the per capita of the patient's stay in the hospital. New York reports an item of some \$25,000 for the transportation of patients. The State hospitals admitted some 3,000 patients, but a good many of them were likely brought by friends. However, if only 2,000 were sent for, the cost would seem to average only some \$12 apiece.

***Transfer of the New York City Insane.***—The transfer of the care of the New York City insane to the State failed to be accomplished. The comments of the various medical journals about the subject show different opinions. The New York Lunacy Commission urged strongly the signature of the order by the Mayor or Governor, but on account of technical difficulties it was refused. The New York *Medical Record* seems to repel the assertion that the city cannot reform its asylum work as well as the State. The only thing seemingly assumed by all is, that the city asylums need reform in some way.

***Hospitals for acute cases only.***—The question of the advisability of a hospital for the acute cases only comes up occasionally for debate. Physicians of the large cities, making a specialty of neurology, would seem especially to have advocated this method, while the hospital officers are quite sure to oppose it, as taking from them all of their interesting cases and all of their basis for a claim to be hospitals and not mere custodial gatherings of people.

It has come about, however, that hospital officers quite commonly admit that acute cases should be separated from the chronic. They admit this much of the contention because of the recognized tendency in a mixed population of acute and chronic cases that the treatment of the acute tends downward toward that of the chronic, rather than that of the chronic upward toward that of the acute. Something like this latter idea is brought out in the following extract from the report of the St. Peter State Hospital for the Insane, by Dr. H. A. Tomlinson, superintendent, which reads as follows:

“Until within the past ten years, all the energies of those who have been connected with the management of hospitals for the insane have been spent in the direction of the housing, clothing and feeding of those committed to their care. This is especially true of those States which have assumed the responsibility for the gratuitous care of all the insane within their borders. Unfortunately, one of the principal results of this change has been an enormous increase in the increment of chronic demented patients, hopelessly incurable, sent not only because they would be better cared for, but also that their families and the county authorities might be relieved of the expense of their maintenance. While



it is a fact that these people are better off and the State does right to maintain them, the result has been to lead the public and the legislature to look at everything connected with the hospitals from the standpoint of the chronic insane. They being a permanent charge, are looked upon from the asylum standpoint, and our appropriations for the whole population are based on the per capita cost of simple maintenance. Until recently the same tendency has shown itself in building, the result being the herding of our patients together in indiscriminate masses. Out of this method of caring for the insane has grown the belief on the part of the public that anyone sent to one of these institutions goes there to stay, and can only be gotten out by process of law; while from this has grown the belief that these institutions are quasi-prisons, where people, having committed offenses against the law on account of insanity, are sent to be restrained of their liberty as a sort of punishment for their misdeeds. I am quite commonly asked on what charges a patient has been committed, and how long he has been sent up for!

However, during the past ten years the medical profession at large, and through it the public, has begun to ask us if we are doing all we can for those placed in our care, and especially in the way of medical treatment. This demand is becoming more and more clamorous and will have to be met sooner or later. Only recently one of our national medical journals (*JOURNAL NERVOUS AND MENTAL DISEASE*, July, 1894) gave up the whole of one issue to an address delivered before the American Medico-Psychological Association, in Philadelphia, by Dr. S. Weir Mitchell, which was a terrible arraignment of our State institutions and their management. This was accompanied by twenty-four letters from prominent neurologists, expressing their opinions and giving their views of how our hospitals ought to be managed. Of course, we think that a great deal of what was said was uninformed criticism, but they all agree in one thing and that is, a unanimous condemnation of the absence of real medical work in our hospitals. I believe that the State which soonest puts its hospitals and their work on a purely medical basis will win as great fame as did Pinel and Tuke, who started the revolution which changed institutions for the insane from prisons to asylums. The carrying out of the hospital idea involves more than the provision of pavilions and infirmary wards. It means the recognition of insanity as a dis-

ease, and the provision of every facility known to modern science for its treatment, especially proper diet, massage, baths, gymnastics and suitable industrial employment. It means, also, the careful systematic physical examination of every patient as an individual, with constant investigation, both at the bedside and in the dead room, of the bodily diseases which accompany insanity and the influence they have in blocking the pathway to recovery, as well as the degree in which they are involved in the production of discomfort and disturbance in those whose mental alienation is permanent. It also means a thorough understanding of the peculiarities, tendencies and capabilities of our patients, so that they may be trained into habits of self-control, cleanliness and usefulness. Then, if, unfortunately, they are permanently deranged, many may still be able to go to their homes and lead useful lives; while those whose condition compels their permanent residence in the hospital may be useful and not unhappy.

It is my belief that we have been all along working at the wrong end of the problem, and increasing our burdens as well as adding to the difficulty of dealing with them, by treating our recent cases the same as we did our chronic cases—herding them together and thanking God when one of them got well in spite of us and his surroundings. Whereas, if we could spend our energies on our recent cases, with proper facilities for treating them, while we kept them separated from the chronic cases, we would cure more of them and eliminate to a large extent those conditions which fill our institutions with patients, who now sit about the wards in idleness, picking their clothing to pieces and soiling their persons. Experience has taught me that it is as easy to inculcate habits of neatness and order as the opposite, if we only begin in time.

To do this work as it should be done would require many changes. First and most important, a separate building for the reception and treatment of new cases adapted for fifty patients of each sex; this building to contain every facility for the treatment of insanity. With this secured the rest would be but the development of details. I know that time is required and that changes from accepted methods can only be made slowly, yet it seems like a truism to say that you cannot do good work with improvised tools or with too few workmen. However, I recognize that we must show by

the work we do that we may be intrusted with greater facilities in order to do more.

The arguments in favor of an acute hospital being supposedly clear, a concise summary of the arguments against selecting one hospital for acute cases alone may be here presented as of value :

1. Patients must be necessarily steadily crowded out of the "acute" and transferred to the "chronic" hospital. These would often feel keenly the hopelessness suggested by the idea of giving up any hope or effort at a certain date, which, indeed, would not be right or pleasant to friends or patients. In this State, for example, of the population at any one time about 60% have been insane for over one year; about 47% of those discharged as "recovered" have been insane more than one year, and about one-half of all admissions have been insane more than one year at the time of their admission. Moreover, the questions of recurrent cases, of periodic cases, of late recoveries, etc., almost make a limit of the curability of cases beyond our power. Either the chronic hospitals would need to be well supplied with means of treatment or great injustice would be done to many cases.

2. The many sicknesses and treatable maladies of the chronic class make provision for like treatment to that among the acute cases, needed simply upon the basis of humane provision.

3. If one hospital were selected for acute insane only in a State, unless it harshly shut out its cases at an early date, it would soon grow to the size of the others and in so far be subject to all of its supposed disadvantages.

4. The distances from home and relatives would be often increased, and the traveling by this and by the many transfers needed would be much increased.

5. The expense would be very heavy for the acute hospital, if small, and not much lessened for the chronic ones. This, of course, should not be a valid argument except as aiding the preceding.

The above is probably a fair resumé of the objections made. By a selection of wards, or as many think preferable—by a separate building the sick, the acute and the actively treatable cases of each large hospital can be kept by themselves, the discouraging and disheartening transfers avoided, and the insidious distinction need not be made.

## Periscope.

### ANATOMICAL.

**Descending Cerebellar Tracts.**—Dr. A. Biedl (*Neurolog. Centralblatt*, 1895, Nos. 10 and 11).

B. performed section of the inferior cerebellar peduncle on young cats, then studied the degeneration of the descending tracts of fibres by Marchi's method.

In the medulla oblongata he found degeneration:

1. In both posterior longitudinal fascicles, more in the left. They derived their degenerated fibres by way of fibræ arcuatæ dorsalis from the injured corpus restiforme. A connection between the posterior longitudinal fascicle and the so called Vorderseitenstrangrest could further be stated by way of radially directed degenerated fibres.

2. In the region which the author calls Vorderseitenstrangrest, and which at the level of the VII. and VIII. roots is situated between the posterior longitudinal bundle and the dorsal border of the superior olive. It is not sharply circumscribed, and gradually passes into the posterior longitudinal fascicles. The connection with the degenerated restiform body is formed by fibræ arcuatæ internæ mediæ partly, but only to a slight extent, also by fibræ arcuatæ externæ. No crossed connection mentioned. The Vorderseitenstrangrest is for the spinal cord what the posterior longitudinal fascicle is for the medulla oblongata.

3. In the so-called Seitenstrangrest, the connection with the degenerated restiform body is given by fibræ arcuatæ externæ, which emanating from the restiform body pass along the lateral border of the ascending V. root, and turning round its ventral border end in the Seitenstrangrest (a small part of them passes further to end in the Vorderstrangrest, as mentioned under No. 2). The Seitenstrangrest occupies the region situated medially and ventrally from the ascending V. root and subst. gelat.

4. In the fibræ arcuatæ internæ which connect the injured restiform body with the inferior olive of the opposite side. Part of these degenerated fibræ arcuatæ, instead of ending here, pass between the olive and pyramids to continue their course as fibræ arcuatæ externæ along the periphery of the crossed side.

5. In several of the roots of the cranial nerves: VI., X., XI., XII. The degeneration found in the VIII. and V. roots cannot be attributed as a consequence of the lesion of the inferior cerebellar peduncles since the nuclei of these nerves had also been involved by the lesion.

In the spinal cord the following degenerations had taken place.

1. In the lateral column. Throughout the whole cord degeneration was found diminishing gradually in caudal direction. The area of degeneration coincides to a great extent with the area of the lateral pyramidal tracts. The author therefore concludes that the area of the lateral pyramidal tracts contain two systems of fibres. I. The system known as the pyramids, which connects the cerebrum with the motor cells of the anterior horns. II. A system which connects the latter directly through the infer. cerebellar peduncle with the cerebellum.

2. In the antero-lateral column of the same partly also of the opposite side. At the higher levels the area of degenerated fibres occupies the antero-medial portion of Gower's column and the mixed lateral column. Diffuse degeneration is found throughout the anterior column also of the opposite side.

The more caudal we pass, the more medial do we find the degenerated area; in the lumbar portion it occupies the ventro-medial corner of the anterior column. Here hardly any degeneration is noted on the opposite side.

The area of degeneration of the antero-lateral column is the direct continuation of the degeneration in the Vorderseitenstrangrest of the medulla oblongata.

3. In fibres of anterior root bundles of the spinal nerves.

To sum up there was, dependent upon lesion of the inferior cerebellar peduncle degeneration in the following tracts:

I. Posterior longitudinal fascicles, some degenerated fibres of which contribute to the formation of roots of cerebral nerves.

II. Vorderseitenstrangrest, continuing into the antero-lateral further caudal into the anterior column of the cord.

III. Seitenstrangrest, continuing into the area which forms part of the lateral pyramidal tracts, which shows that these contain a cerebellar system of fibres.

IV. Fibres of anterior root bundles of spinal nerves.

V. Fibre arcuate internæ which connects the cerebellum by way of the corpus restiforme with the inferior olive of the opposite side.

All these tracts conduct centrifugally having their cells of origin in the cerebellum.

ONTF.

**The Sensible Touch, Temperature and Pain, and Sensory Optic, Acoustic, Olfactory, etc., Conducting Pathways and Centres.**—Jelgersma (*Neurolog. Centralblatt*, 1895, No. 7).

Ramon y Cajal and Lenhossek found that part of the fibres of the posterior roots of the spinal nerves instead of ending in the gray substance of the spinal cord by way of collateral and end branches, enter the anterior horns to become axis-cylinders of the large motor ganglionic cells, these fibres being thus homologous to those of the anterior roots. Probably the conduction in these fibres is centrifugal instead of centripetal as in the rest of the fibres of the posterior roots.

In order to control these results of Cajal and to find whether they also apply to the higher sensory nerves (opticus), the author extirpated one or both eyes of very young doves and examined their central nervous system, after they had become full grown, by Nissl's method. The result was a considerable atrophy of the crossed optic lobe. This atrophy is two-fold: 1. An atrophy of those ganglionic cells of the optic lobe, the protoplasmic processes of which enter in intimate contact with the end trees of opticus fibres. These cells were diminished in size and changed in structure. The author proposes to call this atrophy "functional atrophy."

2. A circumscribed nucleus of the optic lobe (ganglion dorsal optici) has undergone complete destruction, all its ganglionic cells have disappeared, being substituted by an indifferent tissue (numerous nuclei and neurological elements). This kind of atrophy the author proposes to call "organic degeneration."

Functional degeneration (or atrophy) means individual preservation of the nerve cell with alteration of size and structure.

Organic degeneration signifies complete destruction of the ganglionic cell with substitution of the whole by foreign tissue.

Organic degeneration takes place in an injured or diseased neuron and does not surpass its limits.

Partial functional degeneration needs at least two neurons for its development. The first neuron degenerates organically (for instance end trees of sensory root fibres in spinal (after section of posterior fibres) cord), the second neuron, which connects itself with the end of the first neuron, degenerates functionally (for instance the small cells of the substantia gelatinosa Rolandi, etc., which are connected with the aforesaid end trees of posterior root fibres). Sometimes the functional degeneration can extend even over several neurons.

T. then mentions that aside from the complete disappearance of the circumscribed nucleus in the optic lobe a semilunar bundle of fibres which forms a kind of capsule around said nucleus, was atrophied. This bundle must accordingly contain the axiscylinders of the nerve cells of the said nucleus. The fibres of the bundle are evidently homologous to the posterior root fibres of the spinal nerves which Cajal found to take origin from motor ganglionic cells. The grouping of said fibres to a circumscribed bundle in the optic nerve denotes that the latter is higher organized than the posterior roots of spinal nerves. Otherwise there is a complete homology. The spinal ganglion of the spinal nerves is represented by the retina for the optic nerves. The nerve nuclei for the optic nerve are homologous with those ganglionic cells of the spinal cord around which the fibres of the posterior roots spread their end trees. A similar homology with the posterior roots and spinal ganglions of spinal nerves can be found for the acoustic nerve; here the ganglion spinale cochlea corresponds to the spinal ganglion of spinal nerves.

ONUF.

**On the Fine Anatomy of the Optic Roof (tetto optico) of the Teleostei Fish.**—D. Mirto (*Rivista sperimentale di freniatria, etc.*, vol. xxi, 1895, fasc. I.)

M. worked with the method of Golgi. His conclusions are:

I. The superficial fibres of the optic nerve are centripetal; they terminate in the optic roof (tetto optico), a small part of them also in the corpus geniculatum externum by means of the so-called terminal arborisations.

II. Those of the deep (central) optical fibres, which originate from cells of the optic roof, are centrifugal of those which direct themselves towards the corona radiata of Gottsche coming directly from the optic nerve or from the optic roof. Nothing positive can be said neither regarding their nature nor regarding their connections, our knowledge on the general connection of nerve elements not being sufficiently advanced in that direction.

III. The protopasmatic processes in the optic roofs seem to be chiefly apparatus for the storage of luminous stimuli, at least their intimate relation to the terminal arborisations of the superficial optic fibres speaks in favor of this theory.

IV. The protoplasmatic processes of certain cells of the optic roof have such a uniform and fine delicate appearance in their whole course that they can not be distinguished from the nervous processes which give origin to a nerve fibre. It seems that in fish a real differentiation between protoplasmatic and nervous processes is not perfectly established yet.

ONUF.

#### PHYSIOLOGICAL.

**Section of Both Pyramids in Dogs.—Preliminary communication.** T. Starlinger (*Neurolog. Centralblatt*, No. 9).

The technic of the operation consisted in freeing the basilar portion of the occipital bone and trephining it. The pyramids are thus made visible and could be cut through with surety and ease at the point where they come forth from beneath the trapezoid body. The animals first showed slight motor disturbances which gradually vanished, so that two weeks after the operation they did not differ from normal dogs, being just as lively and skillful in their movements. Microscopic examination of the medulla of the operated dogs proved that the pyramids had been completely severed.

The author concludes:

1st. That the pyramidal tracts of dogs have only subordinate importance for locomotion.

2d. That in dogs there must still exist a motor pathway for the

transmission of impulses from the cerebral cortex to the muscles which tract does not have its course within the pyramids. ONT.

## PATHOLOGICAL.

**Facial Hemiatrophy.**—Möbius. *Specielle Pathologie und Therapie*. XI. Band, 1895.

After a short historical sketch the author first considers the subject in a general way. Local trauma seems to be the most frequent cause for its appearance, and especially in younger subjects. The characteristic symptom of this slowly developing disease, is the circumscribed atrophy of the skin on the face. Sometimes confined to a small spot, at other times involving one complete half of the face and even at times extending over to the opposite side, with regard to the depth of the parts involved it also varies. In some the skin is affected. In others the fat layer bones and muscles are involved. The glands of the skin and hair do not escape. Sometimes one-half of the tongue is affected. Besides the circumscribed atrophy other symptoms may alone be present. One of the most frequent is trigeminal neuralgia.

Muscular twitchings, which the author thinks may be due to the neuralgia or active disease of the muscles often show themselves. Occasionally spasm of the muscles of the jaw.

This disease may continue to advanced age.

But sooner or later the atrophy reaches its maximum point and then the condition remains unchanged. The author then goes into a thorough discussion of the symptoms individually.

First: With regard to the nature of the atrophy in the skin. He is inclined to believe that the atrophy in a great many cases slowly advances over the diseased area and have all the characteristics of the simple atrophy of the skin described by Lesser, viz., an advancement with an infiltrated marginal wall clearing behind the atrophied skin. In other cases he supposes this to be merely a simple atrophy and nothing more.

Second: The extension of the atrophy. In the majority of cases only one-half the face is involved. In others both sides of the face. This atrophy may occur, however, on other parts of the body also. Again in those cases where there is merely "Hemiatrophia Facialis," the atrophy may extend over a greater or lesser area of a circumscribed space. The author describes several cases which have come under his own observation with profuse illustrations accompanying.

Thirdly: As a consequence of the atrophy, disfigurement results.

Fourth: Accompanying symptoms

(1). Trigeminal neuralgia. Twitchings are most often noticed in these cases.

(2). Sympathetic disturbances.

(3). Hemiplegia in a case of Pary's. Insanity in Bergson's and Mendel's cases. Epilepsy in a case of Meyer's.

Anatomically there has been discovered in a case of Jolly's a brain sclerosis. With regard to the aetiology the author considers it an exogenous disease. Begins in youth and is more frequent amongst women. The left side more often affected than the right.

The author is very strongly inclined to believe that this disease is due to an external local infection. That this infection may take place either in the region of the tonsils, mucous membranes, or skin. The numerous cases which are cited, and the discussion which the author enters into in order to substantiate this theory should be read in the original.

Pathological anatomy. Here little is to be learned from the autopsies.

Diagnosis.—This should be based upon the circumscribed thinning of the skin. Secondly, that the area is discolored. Thirdly, that it occurs in youth.

The most difficult point is to differentiate between the disease and skleroderma. At times this seems almost impossible. The author is inclined to believe that the two are very closely related.

Course and prognosis.—A case of recovery has up to the present time never been recorded.

With regard to treatment, the author thinks that a prophalactic course in the way of a surgical interference over the infected area may be of the greatest benefit.

WIENER.

**On the Degenerations Following Cerebellar Lesions. Preliminary Communication.**—B. Pelizzi. (*Rivista spirituale di freniatria*, etc.).

P. performed ablation of the medial lobe of the cerebellum on dogs. The consecutive degeneration of tracts of fibres was studied with Marchi's method, with the following results:

1. *Complete degeneration of the superior peduncles of the cerebellum.*

a. Part of the fibres of the retroflexed fascicle of the superior cerebellar peduncles after having crossed the median line at the level of the crossing of the superior cerebellar peduncles turn ventrad and then caudad passing along the raphe to join the medial lemniscus (fillet). They can be traced as a distinct bundle into Gower's tract which they help to form occupying its ventral part.

b. Another part of the fibres after crossing the median line with the other fibres of the superior cerebellar peduncles separate into a superior and inferior bundle. The inferior (ventral) bundle joins the lemniscus and ends in the nucleus of the posterior corpus bigeminum. The superior (dorsal) fascicle passes ventrad of the posterior longitudinal fascicle, and is separated from the inferior one by a stratum of normal fibres; it ends partly in the posterior and partly in the anterior corpus bigeminum.

c. The rest of the fibres (of the superior cerebellar peduncles) pass, after the crossing, towards the red nucleus, part end in the latter, part continue their course through the median and lateral laminae of the thalamus to join the internal capsule and can be traced into the white substance of the central convolutions. The crossing of the superior cerebellar peduncles is probably complete.

2. *Brachia cerebelli ad pontem and pons.*

The degeneration is insignificant in the superficial stratum of the pons, considerable in the deep stratum. After crossing the median line part of the degenerated fibres pass cephalad and join the pyramids of the opposite side; in the pes pedunculi they occupy the inferior (ventral) and external part. Another portion of the crossed degenerated fibres join the lemniscus and ascend towards the posterior corpus bigeminum.

3. *Restiform body.*

Degeneration partial, not of the circumscribed bundle but in small patches throughout the whole restiform body. The degenerated fibres become fibrae arcuatae internae and enter the inferior olive of the opposite side.

4. *Fillet. (Lemniscus).*

Degeneration most intense in the lateral (inferior) fillet in an ascending direction. Most of the degenerated fibres originate from the superior cerebellar peduncles, part come from the corpus trapezoides and pons.

The superior (median) fillet shows degeneration only in its medial and ventral portion; the degenerated fibres originate partly from the superior cerebellar peduncle, partly from the deep pons fibres. Those that occupy the most medial and ventral part of the lemniscus form a circumscribed bundle which probably corresponds to



the fascicle of the fillet to the pes pedunculi and seems to end in the fascicle of Vicq d' Azyr.

5. *Deiter's nucleus.*

It was accidentally injured to such an extent that it was completely degenerated. In spite of this, not the least degeneration in the viii. nerve. From Deiter's nucleus a tract of degenerated fibres could be traced into Gower's tract, another part become fibre arcuate internæ.

6. *Posterior longitudinal bundle.*

It derives its small share of degenerated fibres for the most part from the superior cerebellar peduncle, chiefly from the retroflexed bundle, partly from Deiter's nucleus. A portion of the degenerated fibres viz., those coming from the retroflexed bundle, can be traced downwards to the spinal cord where they become part of Loewenthal's marginal tract.

7. There is also a degeneration of anterior root bundles of the spinal nerves; these degenerated fibres take their origin from Gower's and Loewenthal's tracts.

The other degenerations observed were caused by accidental injury to the nuclei at the floor of the fourth ventricle. ONSIE.

*Contribution to the Ætiology of Dementia Paralytica, with Especial Consideration of Syphilis.*—Houghier (*Illgemeine Zeitschrift für Psychiatrie*, 1894).

The author draws his conclusions with regard to the relation of syphilis to general paresis, in a collection of cases occurring at the Asylum of Lapwik, in Finland, during the years 1875 to 1892.

During these years, 861 males and 659 females were admitted to the asylum. Of these 1,520 patients 107 (98 males and 9 females) suffered from general paresis. The author has carefully tabulated his cases with reference to the active occurrence in both sexes, age, manner of life led by the patients in general, and comes to the following conclusions.

1. That general paresis which is more frequent amongst males than females, is a disease which especially attacks people living in large cities. It does not seem to attack the women of the better class of society.

2. The ætiology seems to be especially in favor of syphilis. The author has shown that this disease (syphilis) plays a very unimportant role in other forms of psychoses when compared to the above.

3. In most cases general paresis occurs between the ages of thirty to forty-five years. It makes its first appearance four to five years after infection with syphilis.

4. The syphilitic symptoms which precede general paresis seem to be of a relatively mild character.

5. In comparison with syphilis, hereditary predisposition and psychological causes; excess in the use of alcohol; sexual excesses and trauma, all held a subordinate position.

6. With regard to the different forms, the maniacal is the most frequent. Next in order is the demented, and finally the melancholic type.

7. Prognosis is unfavorable in all cases. Duration of the disease in 81.8% was four years. In 43.4% two years. Remission were seldom noticed.

8. That general paresis occurring after syphilis, does not show during its course any symptoms which are characteristic of syphilis.

9. No general results were obtained from anti-syphilitic treatment.

10. Autopsies did not show any lesions which were of a syphilitic nature.

WIENER.

CLINICAL.

*A Case of Infantile Giantism (Pedomacrosomia) with Tumor of the Testicle.*—By E. Sacchi. (*Rivista sperimentale di freniatria, etc.*, 1895, Fasc. I.). Family history negative. The patient had measles at the age of 4 years, got entirely cured; no other diseases

or abnormalities up to the age of 5. At this time physical and moral changes became noticeable; a rapid and excessive development of the body, especially of skeleton and muscles; increased growth of the hair, especially on the pubes and face; change of the voice, etc. The left testicle increased considerably in size.

At the age of nine the following condition: Height 4 ft. 9 inches. Weight 44 Kg. (about 90 pounds). Can raise from the ground and carry on his shoulders a weight of 100 pounds. Has 26 teeth. Black, rather thick hair, black beard about 2 inches long, luxuriant growth of hair at the pubes, sternal region and legs also very hairy.

Penis in relaxed state  $3\frac{3}{8}$  in. long,  $3\frac{3}{8}$  in. in circumference. Frequent erections. No ejaculations. Sexual tendencies. No masturbation. Tumor of the left testicle (length four in.), right testicle atrophic, of about  $\frac{2}{3}$  in. diam.

The boy is intelligent, but not precocious. He is of a good-hearted, earnest disposition, does not like to associate with children of his age, prefers to stay at home with his mother. Very conscientious in his school duties, very orderly.

No motor or sensory disturbances of any kind. The tumor of the testicle was removed. It proved to be an epithelial neoplasm caused by coccidii (parasites of the genus *Kariophagus* Steinhaus).

A month after the operation considerable physical and moral changes developed which were most marked 4 months after the operation, when the following condition was present: The dense jet black hairs of the beard have gone and are replaced by fine small blond hair. The hairs at the sternal region and at the legs have disappeared; those of the upper lips and of the pubes have remained. Voice more sharp; it has become an infantile voice. The dimensions of the body (height, circumference of the head, throat, length of the extremities) have not changed. Length and circumference of the penis have gone down to 3 in. Right testicle increased in volume. Erotic tendencies gone. Since the operation no more erections; sexual desire has ceased.

Has become timid, shy, likes playing with children of his age, is more sociable with them. Less obedient to his parents. Still attentive in school. Muscular power considerably diminished (before 98 in dynamometer, now only 75).

Ten months after the operation condition nearly unchanged.

The case is one of infantile (relative) giantism in those cases of which the abnormal growth begins after the age of 14, and absolute giantism, that is a growth surpassing the maximal ordinary stature in the respective population is observed. The cause of the giantism in the case described must be sought in an excess of the nutritive function of the testicle caused by the neoplasma. An analogue is cited in defense of the theory that a neoplasm may increase the function of the organ involved by it.

ONUF.

**Hysteria of Gastro-Intestinal Origin.** (*Gazette des Hôpitaux*). M. Debone analyzed a second paper by M. Clozier, in which he attributed hysteria to gastro-intestinal troubles, and denies that it may be by itself an hereditary malady. It appears in reality that in certain cases the digestive troubles may be the cause of the development of hysteria. But in other cases the digestive ailments are secondary to the hysteria. Finally, there are cases where hysteria exists without digestive disorders either primary or secondary, and in which the influence of heredity is certain.

FREEMAN.

## Book Reviews.

NEUROLOGICAL CONTRIBUTIONS (*Neurologische Beiträge*).  
By Dr. C. J. Möbius. Ambr. Abel (Arthur Miner).  
Leipzig. — Publ.

In these contributions, of which three separate parts are now published, Möbius gives his views on some of the most interesting topics in neurology. In reading the book we are struck first by the clearness of style and the translucency of diction. The descriptions of some of the diseases considered begin with a short and precise definition of the latter. Great stress is laid upon psychological explanation, while anatomy and physiology seem somewhat neglected. One may not agree with all his definitions; that of hysteria, for instance, seems too general, too comprehensive, but one must admit that they are expressed in a clear, decided manner. We find in Möbius a warm advocate of hypnotism, not only as a therapeutic agency, but also as an important factor in explaining psychological phenomena. We further see in him a firm supporter of the syphilis doctrine of locomotor ataxy.

Some of the subjects have been treated on at various dates by the writer, and it is interesting to note how some of his views have been modified in the course of time under the influence of the progress made in certain branches. In 1888 he did not think much of hypnotism as a therapeutic agency; in 1894 he declares hypnotic suggestion to be an important and indispensable remedy.

Passing to a consideration of some of the special features of the contributions, we find that:

Number I. treats of:

- a. The conception of hysteria.
- b. Simulation in nervous diseases caused by accidents.
- c. Mental disturbances following attempts at suicide.
- d. The question whether objections may be raised against the use of hypnotism for therapeutic purposes.
- e. Liberty, accountability and responsibility.
- f. Fundamental views.

Hysteria is considered as a psychosis, the essential feature of which must be sought in the kind of somatic, not of the mental symptoms. In healthy persons conceptions which are associated with extreme sensation of pleasure or dislike effect physical changes (the extremities become paralyzed through fear and terror, sensation of itching when seeing a flea jump, etc.). In hysteria these changes are not only produced much easier, but disturbances may arise which do not occur in healthy persons (for instance, hemianesthesia). This was the view held in 1888. Under the influence partly of the revelations of hypnotism and partly of the works of Charcot, Janet, Breuer and Freud we see his definition undergo a considerable modification. In 1894 it reads as follows. The change characteristic of hysteria consists in that the mental state of the hysteric resembles temporarily or lastingly that of hypnotized individuals. As in the hypnotic state so in hysteria all phenomena (anesthesia, amnesia, paralysis, etc.) are the effect of suggestions, that is of conceptions. All hysterical phenomena are suggestions in form, in contents part of them are not suggested but a pathological reaction upon emotions.

I mentioned before that anatomy and physiology are somewhat neglected by the author. We are sorry not to see any reference made to the neuron theory of Duval and Lépine. These authors explain all phenomena of inhibition, among others hysterical anaesthesia and motor paralysis, as a consequence of the lack of contiguity between the cell ramifications of the neurons; this lack of contiguity they assume to be due to an amoeboid retraction of said ramifications which isolates the neurons in question from the others.

Möbius agrees with Charcot in adding the traumatic neuroses to the group of hysteria. Whether it is advantageous to thus enlarge a group of diseases is questionable; but if we accept Möbius's definition of hysteria, we cannot advance any objection to his classification of the traumatic neuroses. We certainly agree with Möbius in including astasia abasia under the group of hysteria.

In the article on simulation in nervous diseases caused by accidents the author takes a firm stand against Seeligmüller. He thinks that the more the knowledge of certain diseases advances the less will there be talk about "simulation," that is physicians will learn to consider as disease what formerly was called simulation. This is undoubtedly true, but on the other hand by paying too much attention to these complaints we run the risk of increasing the pathological tendency of this class of patients.

The article on the value of electro-therapeutics shows us the merit which Möbius had in sifting this matter. He has called forth a lively discussion on the subject. The result has been very depressing on one side, as it showed how much valuable time has been spent on investigations which yielded but meagre fruit: it was beneficial on the other side, as it has taught to judge the results of electro-therapeutics with less enthusiasm and more criticism, and to first exclude the possibility of psychical influence before attributing a cure to the effects of electricity.

In the chapter of mental disturbances after attempts at suicide we witness an interesting discussion between Möbius and Wagner. On one side (Möbius) the tendency to explain the phenomena on a psychological basis, on the other (Wagner) the seeking after physical causes. Wagner and others observed epileptic seizures and a temporary "retroactive" amnesia in individuals who had attempted suicide by hanging, but could be revived in time. These phenomena, says Wagner, are characteristic and differ from other disturbances (especially mental) observed after other manners of attempts at suicide. They must be looked upon as the consequence of physical injury to the brain caused by anaemia and subsequent retroactive hyperaemia. That the seizures mentioned, he continues, are epileptic is shown by the fact that they can be produced experimentally in animals under the same conditions. Möbius on his part points out that according to certain descriptions of these seizures as observed by other investigators, they were not of an epileptic, but hysterical nature. This being the case he sees no reason why the amnesia should not be equally of purely psychical instead of physical origin. He wants the expression hystero-epilepsy abolished altogether, saying that only the convulsions produced by physical irritation of the brain should be called epileptic. This is a proposition or deduction we can hardly agree upon. The psychical factor in the production of convulsions in cases of idiopathic epilepsy is too strong to allow their interpretation as the effect of purely physical irritation of the brain.

The chapters on "liberty, accountability and responsibility," and on "fundamental views" are of more interest to the psychologist than to the neurologist.

Number II. It treats on akinesia algera, nervousness, especially neurasthenia, and on mental disturbances in chorea.

After reporting three cases of akinesia algera and some mild-

er forms which he proposes to call *dyskinesia algera* M. passes to a discussion of all cases published by other investigators. In reviewing and comparing them with those related by himself he arrives at the following conclusions: All patients, with the exception of Fechner, were degenerates mostly with tainted heritage. The principal exciting cause was over-excitation by preference of the mental sphere, a quantity of function which was too much for the given conditions. Clinically a strict distinction between the essential and the accessory accompanying symptoms must be made. The cardinal symptoms are the pain and other unpleasant sensations dependent upon the function on one side, the limitation of function thus caused on the other side. The pain is of mental origin. A prominent feature is the lessened suggestibility implying the impossibility of being hypnotized. The prognosis is bad; only in one case (reported by Erb) a cure was effected, while in a second (the case of Fechner) a relative cure occurred. Of the others, part ended fatally, part were not observed to the end. The essential symptoms usually aggravate, and in the course of time a tendency to mental diseases in the strict sense of the term—melancholia, mania, paranoia—develops.

M. has widened the conception of the disease. His first definition was: wilful lack of motion on account of the pain which the motion produces. Later we find him speaking of the limitation of function (in a general sense) on account of the pain and other unpleasant sensations which this function causes. By this definition the case of Fechner, and actually also the cases of *atremia* (Nefel) become included in the syndrome of *akinesia algera*. In Fechner's case we find the impossibility of using the eyes as a consequence of the disturbances which this use calls forth. The *atremics* show inability to sit, stand, or walk by reason of the *paræsthesias* following or accompanying these functions.

The histories of the cases of *akinesia algera* reported by M. are rendered in an exact, detailed, and interesting manner, so that in reading them we get a full idea of the character and course of the disease.

The chapter on *neurasthenia* begins with a definition of the same. The symptoms of *neurasthenia* are those of fatigue. *Neurasthenia* is an increased susceptibility to fatigue brought on by activity; the greater the innate disposition is the less needs the sickness producing activity to be. This definition seems indeed very striking and best explains the symptoms. We approve the author's proposition to exclude those cases of increased tendency to fatigue which are the sequels of exogenous diseases or the chronic poisonings (alcoholism), but we, of course, meet with cases in which some exogenous disease, aggravates an innate *neurasthenic* disposition. M. also proposes to exclude the compulsory ideas from the designation of *neurasthenic* symptoms. The phobias have always been classed among the symptoms of *neurasthenia*, and M. therefore makes no objection to considering them as such. The author's proposition concerning the compulsory ideas seems commendable; although frequently observed in *neurasthenics* they are no more typical of the disease than delusions are of melancholia. M. justly calls attention to the frequent combination of *neurasthenic* with hysterical symptoms. The compulsory ideas met with in such cases may just as well be the sequel of the hysterical element of the disease.

Möbius is by no means enthusiastic as to the curability of *neurasthenia*, and who indeed is? Hypnotism usually fails; as a rule it is difficult or impossible to hypnotize these patients. Treatment at watering places is accessible only to the better class of patients; it works discouragingly on him who anxiously watches his pocket book to see whether the improvement increases in proportion with the lessening of the funds. Gymnastics have a depressing influence, because muscular labor is spent and yet no result achieved that could inwardly satisfy the worker by its usefulness.

We cannot but harmonize with the author's conclusion that the best remedy is work of the proper kind, and regulated in such a manner that it will give the patient the conviction that he is a useful member of society, and yet not subject him to the consequences of what, for him, is overexertion. Variations in the kind of work and recreations at appropriate intervals and of proper durations are implied in this plan. This, of course, sounds well theoretically, but practically great obstacles have to be contended against, and M. himself has, as yet, not devised any plan by which this kind of treatment might be made accessible to everybody. The neurasthenic is, of course, very frequently a victim of social disadvantages, which his condition will increase, thus bringing about a *circulus vitiosus*.

The chapter on nervous families gives several genealogical trees which show the strong influence of heredity in the genesis of nervous diseases. The evidence given is convincing. We are glad to see that M. uses the term degeneration discriminately. A protest should indeed be made against the abuse of this expression which Lombroso does not hesitate to apply also to the excessive development of intellectual faculties in calling genius a degeneration-psychosis. M. is right to take a firm stand against this absurd classification. He admits that instability is a prominent feature of genius and that the excessive development of intellectual faculties represents a deviation from the norm which is very dangerous for its bearer. This view of the question meets our full approval.

One chapter is devoted to the discussion of mental disturbances in chorea. Judging from the title we should have expected a more general description of the mental condition of choreatics. We are disappointed in that regard. The author speaks only of the psychoses incidentally observed in this disease, while nothing is said concerning the emotional disturbances observed in such a large number of cases during the whole course of the disease.

#### NUMBER III.

It treats of the etiology of tabes and of locomotor ataxia in women.

The first chapter relates the development of the etiology of tabes. The views of the various authors are communicated at intervals of one or two years. It will thus be seen that from 1880 up to the present the opinions have undergone significant variations. More and more the number of authors who point out the importance of syphilis as an etiological factor has increased. M. himself has been in the ranks of defenders of the syphilis doctrine from the beginning. He holds indeed the most extreme view. While Erb, Strumpell and others still make an allowance for other causes, saying that in a certain percentage of cases previous syphilitic infection could be excluded with certainty, M. considers such infection as a condition *sine qua non* for the development of tabes. He is in favor of Strumpell's theory comparing tabes with post-diphtheritic palsies.

It is interesting to note in what manner M. tries to combat Leyden's arguments against the syphilis doctrine. Leyden mistrusts statistical data, maintaining that it is known what errors such statistics are subjected to. M. treats this objection in a satirical way, saying that whatever errors may occur consist in that the patients conceal their syphilitic infection or are ignorant of it. This is a rather bold assertion. It is evident from M.'s own reports that signs of syphilis are detected in a minimal number of tabetics only. Direct evidence of foregone infection can consequently not be had. The report of the patient is all we have to rely upon in most cases. Is there really never any possibility of misinterpreting such reports? One should think there is, and it seems that the following case gives a rather striking illustration for this possibility.

In examining a baby of about six months of age I found some symptoms which made me suspect hereditary syphilis. We all consider repeated abortion as one of the strongest proofs of syphilis. I asked the mother in that regard. The answer was that she had aborted once. Afterwards my questions became quite direct, but I could not obtain any further evidence of syphilis in the parents of the baby. Later inquiry from another source made it very probable that she had not aborted once but twice, that both times, however, she had brought on the miscarriage by artificial means. This, of course, does not prove anything against the possibility of syphilis, but it demonstrates that in this case the abortions had absolutely no diagnostic value.

If we add that frequently lack of time does not permit a closer inquiry, we may feel satisfied that often the anamnestic reports are misinterpreted in favor of Lues. Is it not peculiar that in 1880 Eulenburg found only one case of locomotor ataxy out of 149 in which syphilis had to be looked upon as the most probable cause and that in 1885 the same author stated previous syphilitic infection in 39 out of 166 tabetics?

The strongest argument in favor of M.'s assertion that syphilis is a *conditio sine qua non* for the acquisition of locomotor ataxia lies in the fact (at least M. reports it as a fact and has not been contradicted) that tabes was never observed yet in virgins. But the value even of this proof must not be overestimated. M. himself admits that most frequently puerperal processes seemed to have hastened, if not brought on, the development of tabes in women. In two cases out of eighteen the first symptoms appeared after a normal confinement, in one after abortion with hemorrhage while exacerbation of the disease occurred after a confinement in one case, after a difficult confinement with strong hemorrhage in another. In a sixth case hemorrhages caused by a myoma of the uterus had brought on considerable impairment. Be it mentioned besides that of the said eighteen female tabetics only two had not been gravidæ, one had suffered abortion, fifteen had born children at term. Some of these fifteen had had abortions. The influence of puerperal processes becomes thus rather evident. Moreover, M. himself does not deny that severe loss of blood is an important causative (as an exciting cause) factor. Virgins are, as a rule, much less exposed to uterine hemorrhage, which in women is perhaps the most frequent source of loss of blood.

Edinger's new theory is quoted by M., but he thinks that E. overrates the importance of function in the causation of the diseases in question, especially of locomotor ataxia. Yet M. does not give any satisfactory explanation why relatively so small a percentage of syphilitics become tabetic, and what brings on the disease in these cases. It further astonishes us that M., although quoting the ergot-tabes, makes no mention of the pseudo-tabes of diabetics and anemics.

It is not the intention of the reviewer to convey the impression that syphilis is not a very important factor in the causation of locomotor ataxia, on the contrary. To contend against the extremeness and exclusiveness of M.'s views seems, however, necessary.

B. ONUF.

THE ART OF MASSAGE. By J. H. Kellogg, M.D. Modern Medicine Publishing Co., Battle Creek, Mich.

This treatise on the practice of massage, by the superintendent of the Battle Creek Sanitarium, belongs to a bad class of books, although it has certain virtues of its own. It is "intended for the use of medical

students and student nurses," but it is as far over the heads of such persons in much of its language as it is below the requirements of any one who should wish to acquire the "scientific knowledge of the art and science of massage," which it professes to give.

The author states very properly that to learn this art "personal instruction is absolutely indispensable," and then proceeds to the most elaborate, and, we should fear, incomprehensibly written instructions for the minutest movements. A much better plan would have been to live up to the statement by briefly describing the several movements, leaving the minuter experiences to be taught only as they can be taught, by demonstration and practice—a plan pursued by Kleen in his recent work on the same subject, a book in which the practical parts are at least as practical, and the scientific appreciation of the aims of and indications for massage far superior to the treatment of the same matter by Dr. Kellogg. The author makes the mistake of hair-splitting in the sub-dividing of the several movements. For instance, it is mere folly to call "touch" a massage procedure; nor is the absurdity lessened by saying that it "it is not an ordinary touch . . . but a skilled or professional touch." This is the usual error of the common masseur, who would have every possible kind of handling to which the body may be subjected classified as some kind of massage, but it is not what one would expect from a physician. The description of the "physiological effects of simple touch" only makes matters worse—" [1] Elevation of the temperature of a part by the communication of animal heat. [2] A subtle influence on the nervous system—the so-called hypnotic effect. . . . [3] It is possible that certain electrical effects may result from simple contact." Bosh is a mild term to apply to these presentations of the value of "simple touch." If "simple touch" is massage, spanking is a scientific procedure, and the varieties of it, as "the simple hand," "the common shingle," "the elaborate buckle-end of a strap," are susceptible of careful classification.

Fortunately not all the book is on this plane, although there are some other suggestions for treatment calculated to make the judicious weep. The description of *pétrissage* could scarcely be bettered, and the whole section on the uses and modes of application of abdominal massage is exceedingly good.

It is a pity that Dr. Kellogg should have chosen to invent a number of names for the different movements when the French ones are already in such universal use among masseurs, and certainly the new titles have no advantage of euphony. "Chucking," for instance, is a new and not very pretty name for a very slight variation in the ordinary two-handed application of *pétrissage*.

One of the most undesirable teachings in the book is the instruction several times repeated for the use of centrifugal stroking, a movement which has little or no value, and for which the indications are of the utmost rarity.

The illustrations are pleasanter to look upon than is usual in massage books, and probably no more useless than usual.

JOHN K. MITCHELL.



THE  
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**Nervous and Mental Disease.**

AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-first Annual Meeting, held in Boston, June 5, 6  
and 7, 1895.*

*(Continued.)*

*Morning Session, June 7.*

THE DIAGNOSIS OF PACHYMENINGITIS  
INTERNA HÆMORRHAGICA.

BY WILLIAM N. BULLARD, M.D.

ABSTRACT.

Dr. BULLARD does not propose to enter into any discussion of the etiology or pathology of this affection and does not intend by the use of the name pachymeningitis interna hæmorrhagica to imply any pathological theory.

This affection may for the sake of convenience be divided into three forms differing in their etiology and in the conditions under which they occur. These are I. The traumatic cases in adults. II. Cases appearing in infancy and generally due to continued or violent pressure against the cranium during birth. III. The apparently spontaneous, idiopathic, non-traumatic form occurring usually in adult life and in connection with chronic cerebral disease, insanity, paralytic dementia or alcoholism.

It is the latter form (III.) only which is considered in this paper. In cases where the hæmorrhage is very

slight the diagnosis is at present impossible. Where the patient is insane or demented the symptoms of hæmorrhage may be so masked that they do not attract the attention of the physician.

In the more evident and more severe cases the symptoms on which we rely for diagnosis are those of intracranial hæmorrhage. When apoplectic symptoms occur in paralytic demented or in the chronic insane, we should bear in mind the probability that the hæmorrhage may be of this character. In chronic alcoholics it may also occur, and is then to be differentiated from acute œdema of the brain. Subdural hæmorrhages in the adult are to be distinguished from other intracranial hæmorrhages also by the character of the symptoms. In many cases their onset is more gradual than in the ordinary forms of intracranial hæmorrhage and the irritative stage lasts unusually long. The irritative symptoms are prominent. General epileptiform convulsions and localized convulsive movements are apt to occur.

The peculiar rigidity localized in one limb when occurring in connection with symptoms of hæmorrhage, and where no affection like tubercular meningitis exists, is almost pathognomonic. The absence of involvement of the cranial nerves is in favor of this form of trouble.

A case of this affection was reported occurring in a man sixty-five years old, in which operation was performed with some temporary benefit, but too late to save the life of the patient.

#### DISCUSSION.

Dr. TOMLINSON, of St. Peter.—With regard to the pathology. In the first place, I think the term pachymeningitis is a misnomer, because I do not believe the condition is inflammatory primarily at all. As far as my personal observation goes among the insane, these hæmorrhages are always preceded by degenerative changes in the arteries in the dura, and I have seen them varying from little spots, the size of a ten cent piece, to others that spread over the whole dura, covering not only the surface of the brain, but the membrane at the base as well, and one case in particular, a case of general paralysis in which, as I recall it now, there were four different layers of membrane in different stages of organization, the one nearest the dura decolorized and the last was pure red. In this case the cause of death

was, finally, a much larger hæmorrhage in the temporal region on both sides. Now, in the first place, there is no sign of active inflammation in the dura and in a great many of them no material thickening, but there is a marked change in the blood vessels, and the apparent morbid change seems to be hæmorrhage from small vessels which is persistent, and the blood gradually oozes out and spreads over the surface of the dura. The commonest seat is the vertex, in the insane, because there they are most likely to receive blows and other traumata. How the idea with regard to this being inflammatory originated, I do not know. I know of one or two papers having been written upon this subject from this same standpoint, the fact of this not being an inflammatory condition, but primarily a hæmorrhage due to the rupture of degenerated arterial walls, and if there is any inflammation following, it is a secondary condition. So far as the clinical manifestations are concerned, they occur in the insane just as described by Dr. Bullard, except that we are not often able to make the diagnosis unless the hæmorrhage is a gross one.

Dr. FISHER, of New York.—I should like to ask whether Dr. Bullard believes that the blood comes from the new-formed blood-vessels or from the blood-vessels of the pia mater. Some authorities think it is from those thin-walled blood-vessels, the result of inflammatory conditions. Sometime ago this subject came up in the Pathological Society of New York. I had a number of specimens of pachymeningitis hæmorrhagica, and Dr. Northrup took some of those cases and made a careful examination and found this new growth tissue which evidently was inflammatory in character, and the blood evidently came from the thin-walled new blood-vessels, not from the regular arterial degeneration which we generally do find to be sure in those cases of pachymeningitis.

Dr. TOMLINSON, of St. Peter.—The reason I spoke so positively is because I have seen this hæmorrhage, that is a layer of blood spread over the dura, before there were any blood-vessels organized in it, and in one individual I saw the membrane in three different stages of development, first, the hæmorrhage in one place; next, the organized membrane with the blood-vessels, and finally, the decolorized membrane; that is, the coloring matter of the blood having disappeared and nothing remaining but a transparent membrane. What made

me observe this so particularly was because it was at the time that this article of which I spoke appeared in the *American Journal of Medical Sciences* making this claim, and on that account I studied the change very carefully in a case of senile dementia, and the three conditions existed over different portions of the subdural surface and in the form I have described.

Dr. FISHER, of New York.—I should like to ask what are the special indications for operation, the symptoms that actually decide on an operation. We find in some of these cases of pachymeningitis hæmorrhagica that the blood is absorbed.

Dr. BULLARD, of Boston.—I think as a rule the symptoms that would call for operation would be the same as those in other cases of traumatic hæmorrhage, those in which the symptoms suggest compression, in which we have severe paralyses following the effusion of blood, those in which the patient's life is in any way threatened, and then according to the judgment of the operator all those cases in which there is moderate hæmorrhage and in which he felt that it was, although not absolutely necessary to remove the blood, yet that on the whole the operation not being a severe one it would be safer for the patient to remove it rather than trust to its being reabsorbed.

In regard to the pathology, it was Virchow who first advocated the inflammatory view as is well-known. The other view was upheld by the English surgeons. The gentleman to whom Dr. Tomlinson refers is Wigglesworth. Other persons like Dr. Wigglesworth have suggested that the blood comes from the vessels of the pia. The evidence seems in favor of non-inflammatory origin, but very acute observers are still in favor of the inflammatory origin. Dr. Osler still holds to that view. Dr. Fitz recently expressed the opinion that it is in many cases inflammatory. Although I myself am inclined to favor the non-inflammatory view, I think the question is still to be considered subjudice.

Dr. STARR.—Dr. Delafield teaches the inflammatory view.

Dr. TOMLINSON.—I only meant in the insane. The other case I know nothing about.

## A CASE OF INCIPIENT LOCOMOTOR ATAXIA AND MONOPLÉGIA FROM FOCAL LESION OF THE INTERNAL CAPSULE IN THE SAME PATIENT.

BY DR. MORTON PRINCE, OF BOSTON.

Dr. MORTON PRINCE showed sections of the cord from a case of incipient tabes associated with a monoplegia, the latter being due to a focal lesion (softening) of the internal capsule. There was a history of syphilis. The diagnosis of tabes was based during life chiefly on the presence of laryngeal paralysis and Argyll Robertson pupil, as there was no ataxia, no sensory symptoms (pains or anæsthesia), and the knee-jerk was present. Later, slight strabismus was observed. The sphincters were unaffected. Tracheotomy was necessitated on account of the danger imminent from the laryngeal paralysis. Death was accidental. In the sections of the cord the posterior columns and the posterior nerve roots were moderately but plainly degenerated. The monoplegia was largely limited to the hand (right), which was almost absolutely paralyzed. The arm was slightly affected. Sensation in the hand and arm was unimpaired. At the autopsy a thrombosis from endarteritis had caused a focal softening of the internal capsule, but the exact position in the capsule was not determined.

## A CASE OF LANDRY'S PARALYSIS WITH AUTOPSY.

Dr. PRINCE also showed sections of nerves from the brachial plexus taken from a case of Landry's paralysis.

The paralysis had developed suddenly, the patient having been found in bed paralyzed in both arms and legs about one hour after a time when he was known to have had the use of his limbs. Sensation and the sphincters were unaffected; but the reflexes were abolished. Death took place twelve hours after the onset of the paralysis, probably from extension to the respiratory centres.

At the autopsy, and examined microscopically, the cord was normal. The nerves from the brachial plexus prepared by the Weigert method also showed nothing abnormal. The sections were shown in view of the attention which has of late been directed toward the presence of neuritis in Landry's paralysis.

In this case the early occurrence of death would probably account for the absence of secondary anatomical changes. The clinical facts and the negative post-mortem findings harmonized with the toxic theory of the disease. There was an alcoholic history.

### DISCUSSION.

Dr. STARR, of New York.—Did this man have high temperature and the general appearance usually recognized under the term septic?

Dr. PRINCE.—He did not.

Dr. STARR.—I have recently seen a very sad case of Landry's paralysis, in which within four days a man in apparently perfect health went through all the stages of rapid paralysis ascending from below upward, and finally died, and in that case the temperature after the second day was continually above  $103^{\circ}$ , and his whole appearance even from the second day on which I saw him, was that of extreme sepsis. He had that disagreeable color, coated tongue and general septic look that gave me the notion that the man was dying of some

acute septic process. Unfortunately I did not get an autopsy. It has seemed to me that reason why we find no lesions is because the process is so rapid that the changes are not well enough developed to detect them with our present methods of staining.

Dr. PRINCE, of Boston.—I saw a case of Landry's paralysis in a man who undoubtedly had some form of septicæmia. He was brought to the hospital with a false passage in his urethra caused by attempted catheterization. A typical Landry's paralysis developed. He died in a few days. There was reason to believe there was some septicæmia from this false passage.

A CONTRIBUTION TO THE PATHOLOGY AND MORBID ANATOMY OF AMYOTROPHIC LATERAL SCLEROSIS, WITH A REPORT OF TWO CASES WITH AUTOPSY, IN ONE OF WHICH, OBSERVED BY DR. C. L. DANA, A TERMINAL TUBERCULAR CERVICAL MYELITIS AND PERFORATING NECROSIS OF THE DORSAL CORD WAS FOUND.

BY DR. JOSEPH COLLINS, OF NEW YORK.

ABSTRACT.

After referring to the moderately constant clinical picture of amyotrophic lateral sclerosis, and the variable pathological conditions, on which they have been founded, the author referred to the fact that the number of cases in the literature, which were well substantiated by autopsy, were fewer in number than one might be inclined to think; a moderately thorough search of the literature revealing only seventy-two cases, which satisfied these conditions. Reference was also made to the different views held by the followers of Charcot and Erb on the one hand, and Gowers, Leyden and others on the other, as to whether the disease is primarily of the pyramids with a secondary involvement of the anterior horns, or whether it is but a variation from the common form of progressive muscular atrophy. The opinion was advanced that, although there was probably more evidence in favor of the former view, this question cannot be settled until a very much larger number of cases, which have been verified by careful histological studies, have been recorded.

The history of two carefully observed cases was then given. The first case was a male, 33 years old. Russian. No pertinent family or personal history. No attributable cause for illness, except overwork and conditions which deprave nutrition, viz. poor and insufficient food, unwholesome and crowded surroundings, and excessive cigarette smoking.

First symptoms in summer of 1893, consisting of



pain and stiffness in neck, headache, vertigo; later stiffness in face muscles, loss of dexterity in fingers of right hand, and later in left; inability to open mouth, dysarthria, dysphagia, nasal twang, increasing difficulty in locomotion on account of stiffness and spasticity.

Examination.—Masked face, considerable atrophy of hands, thenar, hypothenar eminences and interosseous spaces, some atrophy of forearm, more marked on right than left side. Spasticity of upper extremities. Spontaneous and stimulatable fibrillary twitchings. Tendon reflexes all exaggerated. Occipital phenomenon, jaw jerk, triceps reflex all pronounced. Tenderness when atrophied muscles subjected to light squeezing. Cannot walk unless supported. Hyperæsthesia of lower extremities to all forms of stimulation, except faradic. Strong faradic current, such as painful to normal individual, agreeable to him. No real palate pharyngeal anæsthesia, and palate reflex preserved. Special senses normal. No vesical disturbance. Physical condition good. Has peculiar inhibition of emotional reflex, spontaneous and uncontrollable waves of smiling and attempts at laughter. Electrical examination, diminution of faradic irritability, quantitative lessening of galvanic. Unwieldiness of tongue, difficulty in swallowing, respiration weak, pulse rapid. Rather rapid progression of all the symptoms, and after few months the lower extremities began to atrophy. All the symptoms became extremely aggravated, and the patient became bedridden, and death occurred from failing heart and pulmonary oedema.

Autopsy showed no marked deviation from the ordinary in the outward appearances of the spinal cord and brain. The anterior root nerves and hypoglossal nerves were small. On cutting the cervical cord across there was seen a reddish brown discoloration of the anterior horns from the third to the sixth segments, and the consistency of this area was lessened. The entire motor system from cortex to periphery prepared for examination by the methods of Marchi and Algeri, and the Nissl, the Weigert, carmine, nigrosine, hæmatoxylin dyes, etc.

In the spinal cord massive atrophy of the ganglionic cells throughout the cord; best preservation of the cells in the lumbar region, but great diminution of numbers and degeneration of cells here. Degeneration of the crossed and direct pyramidal tracts; few fibres were left undegenerated. Marchi stain shows that the de-

generative process has reached its completion. Throughout the cord, but especially in cervical and dorsal region, evidences of excessive vascularity in shape of large thickened blood-vessels, especially in the gray matter and spaces from which vessels have dropped. In the cervical cord corresponding to the place of apparent softening in the recent state was found great disorganization of the ground substance of the anterior horns.

In the medulla degeneration of the hypoglossal nucleus throughout its entire extent, with the exception of the extreme cephalad end. Slight degeneration in the tenth and common vago-accessorio-glosso-pharyngeal nucleus. No degeneration of the pyramids of the medulla. Root of the 12th nerve small and delicate.

Marchi or Weigert method does not reveal any degeneration in the motor tract from the end of the medulla to the cortex. Nissl's stain shows the cells of the cortex to be normal.

CASE2.—Male, 48, piano maker. Denies alcoholic habits and syphilis, and is of good family and personal history. In October, 1893, first noticed a temporary difficulty and indistinctness of speech and later weakness in left arm; this latter became gradually worse; speech difficulty bettered, but recurred later and remained permanent. Later legs became weaker, and had difficulty in walking. In January, 1894, speech trouble was very slight and arm trouble most prominent. Examination showed that the hand muscles were most affected; the arm and shoulder muscle next. Unable to flex or extend fingers except feebly. Shoulder, arm and forearm muscles all show decided atrophy, but this did not affect any particular physiological groups. No contractures; no fibrillary contractions. Tendon reflexes all greatly exaggerated. No sensory disturbances. Some rigidity of the elbow and shoulder. No disturbances of special senses. Peculiar spasmodic movements of the lips, tongue and jaws; more particularly of tongue; movements as though he were licking lips, or chewing movements—these all beyond his control. Speech disturbance appeared as though due to stiffness and spasm of the tongue. Speech not exactly syllabic. June 15th, 1894—chilliness, fever and weakness; afterward continual elevated temperature, 102 to 104; paralysis in arms and legs increased, little pain in back, slight anæsthesia of legs and arms for short time preceeding death; ten days later unable to speak or swallow.

The spinal cord on section in the fresh state was found to be soft in the neighborhood of the upper dorsal region, and on cutting it down through the dorsal region a spot of white softening could be traced along through the posterior columns. This perforating softened area grew smaller and ended in neighborhood of the lumbar cord. A piece of cord from the cervical region placed in alcohol and culture made from it revealed the presence of the tubercle bacillus in considerable numbers. In cervical region on account of the terminal tubercular myelitis sections, hardened in Müller's fluid and prepared for hæmatoxylin, were very unsatisfactory, but degeneration in the crossed and direct pyramidal tracts with almost complete destruction of the anterior horns could be made out. In the dorsal region there was sclerosis of crossed pyramidal tracts, slight sclerosis of ascending cerebellar tract, diffusely distributed dilated blood-vessels, some with very much thickened walls. Extensive atrophy of cells of anterior horns. In the dorsal region there was found an artificial displacement of the columns of Clarke on one side, so that the column was in juxtaposition with the column of other side. Some degeneration in lumbar and sacral regions as in dorsal, but in lesser degree. In medulla atrophic changes in the nucleus of the 12th nerve. Hæmorrhage of ancient date in dorsal region.

The findings of these two cases were then analyzed and commented on in detail.

The first case would seem to give testimony in favor of the view enunciated by Charcot, inasmuch as the Marchi method showed that the degeneration was complete and apparently of somewhat ancient date, whereas the changes in the gray matter were still going on. The intimate and dependent relationship existing between the striking vascular changes and the extensive degenerations, were then discussed, particularly in reference to what may be called the neuron theory. Specimens and drawings of the pathological changes described were exhibited.

Dr. PUTNAM, of Boston.—There is one point of physiological interest which Dr. Collins referred to, on which I should like to say a single word and that is the emotional characteristic which this patient exhibited in connection with these lesions in the medulla. I suppose we have all noticed the extreme emotional tendency of patients with glioma in that region. I certainly have

seen this in several instances, and Oppenheim has recently called attention to the emotional loss of control in patients with bulbar paralysis. I think he referred more especially to pseudo-bulbar paralysis, but at any rate in connection with a disturbance in the action of those parts which are related to emotional display, the emotional sensations, feelings become prominent, and in connection with the recent psychological views advanced by Prof. James and others, I think, that point is one of considerable interest.

## REPORT OF A CASE OF PERONEAL MUSCULAR ATROPHY, WITH AUTOPSY.

BY WM. C. KRAUSS, M.D., OF BUFFALO.

### ABSTRACT.

He reported the case of a man, seventy-eight years of age, who, at the age of eighteen, began to notice a weakness of the leg muscles. This weakness was accompanied by atrophy in the peroneal and hamstring muscles of both legs and extended to the muscles of the thigh, the left thigh being much more affected than the right. Double club-foot of the *pes varus* variety resulted, and also a double *genu valgum*. The tendon reflexes were absent on the left side, and also on the right, with the exception of the knee-jerk. Fibrillary contractions and sensory disturbances were entirely wanting. There were present lordosis and scoliosis of the lumbosacral region. The muscles of the upper extremities were unaffected. The patient gave no history of any infantile disease or of poliomyelitis, and ascribed the affection to an accident. The patient died from uremia. Microscopic examination of the spinal cord revealed atrophy of the anterior horns, especially on the left side, extending from the caudal part of the thoracic region to the conus medullaris. The multipolar cells of the antero-lateral group were visibly affected, in places having disappeared, in others undergone atrophy or degeneration, while the median group was less affected. The white matter was somewhat sclerosed, particularly in the antero-lateral and posterior columns, but not more than the age of the patient would warrant.

### DISCUSSION.

Dr. JOSEPH COLLINS, of New York.—The question which Dr. Krauss has brought up is such an important one that I feel we would be doing ourselves an injustice were we to pass it without discussion. He has asked us either to support him, and therefore take up the ques-

tion which Hoffman has apparently spoken on authoritatively, or deny what the latter has said. Now the fact that Dr. Krauss is reporting a case of peroneal muscular atrophy in a patient of seventy, when all the cases heretofore have been under twenty, is something akin to marvelous, and if we are going to accept this case as one of peroneal muscular atrophy, it means a radical change in our ideas of the time of occurrence of the disease; but I for one am not quite ready to do that. In Dr. Krauss' case, so far as I can see, there are changes in the spinal cord, which are rather significant of ancient poliomyelitis; but I might say without farther ado, that I do not wish to speak too positively or give my opinion unreservedly in the case. Hoffmann, as you know very well, has established this form of progressive muscular atrophy without any changes in the spinal cord, while Sachs, who did some of the best and some of the earliest work in this disease, described changes in the spinal cord in these leg types, which has been confirmed by Dr. Hammond. Sachs has, however, since that time recanted his views, or rather his statements about one of his cases which seemed to show disease of the cord. In Dr. Krauss' case there is to be seen with the greatest ease, even with the naked eye a shrinkage of the entire cornu of the gray matter. There are to be seen, I think, some slight vascular changes in the blood-vessels in the shape of ancient thickenings. There is, as he has told us, to be seen a sclerosis of the ganglionic cells, and many of the cells show a condition of necrocytosis.

Now, these are changes which are, I believe, apart from the degenerative changes secondary to senility. The change in contour of the anterior horns and the shrinkage point to a previous affection, inflammatory, of these parts. If these were not so apparent in Dr. Krauss' specimens, I should feel more inclined to think, considering that the cells of the anterior horn, the anterior root nerves and the peripheral nerves are one unit, that the changes in the cord were but a part of a change which involved the entire peripheral motor neuron, a change most pronounced at the peripheral terminal distribution of that neuron.

One of the most difficult differential diagnoses to make, and one which has been insisted upon by Hoffman, Erb, and Sachs is that between progressive muscular atrophy of the peroneal type and poliomyelitis. Until Dr. Krauss establishes the fact that this patient

did not have a poliomyelitis, and there are two or three things in the clinical history which, unfortunately, are lacking, we cannot accept this case as a true case of the peroneal type of progressive muscular atrophy.

Dr. KRAUSS, of Buffalo.—I tried as hard as I could to make an electrical examination of this patient, but he was in a religious institution and refused point blank. I also tried to have better photographs made of the man, but he also refused. He was paying his way in the institution and did not wish to be encroached upon. The only feature in the case in the symptomatology which does not agree with the peroneal type of atrophy as described is the hereditary part. There is no heredity, it is not a family disease in this case. I think the other symptoms tally accurately with the descriptions Dr. Sachs gave this Society five years ago. As far as I could learn, I could not obtain any of the symptoms of either acute or chronic poliomyelitis, and I was hoping that Dr. Sachs would be present at this meeting so as to throw the diagnosis overboard or accept it.

## TWO CASES OF WRITER'S CRAMP.

BY DR. JAMES W. PUTNAM, OF BUFFALO.

TELEGRAPHERS have frequently been victims of cramp, and some cases have been reported as suffering from both telegrapher's and writer's cramp. This is explained by the fact that the same sets of muscles are largely used in both operations, and also by the fact that most operators write almost as much as they send.

A case of this came under my care in 1892.

T. D. F., aged 30, railroad telegraph operator, nervous temperament, gave me the following history: Both parents were nervous, the father a great user of tobacco from boyhood, and also used liquor freely; brothers and sisters all nervous.

At twenty-two patient began telegraphing. He was never a rapid sender, just a fair operator. His sending was called heavy and firm.

In 1886 he noticed that his sending was failing. He made the characters of the Morse alphabet all right, but it did not carry, the operator at the other end called it light.

He could not say he had cramp, but it was a weakness of the wrist.

A peculiar part of it was that at any time he could make the characters that commenced with a dash all right. The characters that commenced with a dot he could not make.

Soon after this he noticed that while writing he had to grasp the pen tightly to keep his hand from "scattering;" also experienced some pain in the wrist and cramp.

In 1887 he found it impossible to do any telegraphing or writing with his right hand. Nor did he try to do so for the next two years as his symptoms had grown so much worse.

As soon as he grasped a pen or pencil the arm would kink or contract and cause a contraction of the whole of the right side.

The cramp affected the neck muscles so that the head



was twisted around to the right and would see-saw as he described it about like opening and shutting a gate. Gradually these symptoms increased until working with the left hand became equally difficult.

The head would pull around to the right and the eyes would be turned to the left in an attempt to see what he was writing. He continued using his left hand, although he experienced great pain in the head and eyes, whenever he wrote, looking at the paper.

If he looked out of the window, however, he could write without any pain in the head or contraction of the neck muscles. The instant that he looked at his hand the pain returned.

At this time, 1889, he gave up trying to write, and commenced dictating. This was only practical for a short time, for he found when he watched the clerk writing the head pulled to the right and with as great pain as when he wrote himself.

The symptoms increased to the extent that he could not walk into an office and see any person writing without immediately having pain in the head and cramp in the neck. At night if he thought of writing the same cramp would seize him so that he could not keep his head from turning on the pillow.

With other movements of the right hand, such as spreading butter on bread, strapping a razor, the same contraction occurred.

In this condition he consulted me.

His previous treatment had been thorough; it included prolonged rest, different drugs, electricity for several months. Examination of his arms showed no anæsthesia, no tenderness along the nerve trunks. Some tenderness of Erb's point.

There were none of the ordinary stigmata of hysteria, such as anæsthetic and hyperæsthetic zones. Conjunctivæ and pharynx were sensitive; his reflexes were normal. He was then asked to write. Taking the pencil in the ordinary way, he wrote two letters when the muscles of the right arm became rigid, the left sterno-cleido mastoid was thrown into a strong tonic spasm; also the trapezius drawing the head way over to the right. The eyeballs also rolled up so that the cornea were almost out of sight. The patient remained in the following position while the pencil was near to the paper: fingers firmly clenching the pencil; arm rigid; head turned to an extreme degree to the right; chin a little elevated; eyeballs rolled up.

When I told him that would do, he relaxed immediately. When he tried to write with the left hand the attempt was more successful. The head only turned to a slight degree to the right, so that he was able to see the paper.

He was sent to Dr. Lucien Howe, of the Buffalo, who examined his eyes for me, and who reported normal vision and muscular balance.

In his case the symptoms of cramp at the mere thought of writing or witnessing of writing, were so clearly psychic in their nature that suggestive treatment was instituted.

I hypnotized him regularly three times a week for a month. This was done easily by means of fixation of vision upon a bright object. This would sometimes be my watch and sometimes a magnet.

When he was asleep no suggestions of any kind were given him, except such as would have direct relation to his symptoms and their removal. More than one new suggestion was never given.

For example, I always impressed upon him that the cramping of the neck muscles would be less when he attempted writing than it was before.

After leaving him in hypnotic sleep for from five to ten minutes he was awakened. He always attempted writing before leaving the office.

Toward the end of his month's treatment he had improved to this extent, that his neck did not cramp when he wrote with his left hand, and cramped only to a slight degree when he wrote with his right hand, if he wrote in my office and by my direction. His improvement encouraged us both, until he reported that his cramp was as bad as ever when he attempted to write away from my office.

The treatment beside hypnotism was galvanism of the affected muscles, rythmical, gymnastics and deep injections of atropia,  $\frac{1}{20}$  gr., into the affected neck muscles.

He improved to some extent, being able to write his name, and occasionally several lines with only a slight cramping of the muscles of the arm and no cramping of the neck muscles, when he wrote in my office. He left me in this condition, and as a final psychic therapeutic measure, I told him that in a year he would be all right, but not to attempt writing till that time had elapsed.

I did not see or hear of him again till March, 1894,

when he called on me and finished his clinical history for me. He went away greatly encouraged, as he had been able to write in my office without the cramp of neck muscles. He continued his exercises and avoided writing for six months; this made a total cessation of two and one-half years. He then thought he would try writing, this was April, 1893, and since that time he has gradually increased his writing, until now he does his work at Kinzua, from eight to ten hours a day without cramp.

The second case illustrates the fact that writer's cramp may develop without being brought on by excessive writing.

Dr. H., a dentist, nervous temperament. Has practiced his profession very steadily for over fifteen years. His work has been almost exclusively filling of teeth, working at this several hours a day. In doing this he grasps a small steel instrument firmly with the thumb, index and middle fingers, about as we hold a pen. Then resting his hand on the face he makes continuously and in rapid succession a series of blows on the gold fillings. This action is, of course, done by the forcible contractions of the flexors of the hand, with the wrist as a fixed point.

As a result of this he has found that often at night his forearm muscles ached.

He has never found, however, that this has interfered with his work.

About three years ago he experienced difficulty in writing, because of a cramp in his thumb and index finger.

This increased until, when he consulted me, he was just able to write his name.

This case is interesting, in that it is a case of writer's cramp not due to writing, but to an overstrain of the muscles used in writing, or rather used in holding the pen.

This cramp does not interfere with his work at all, on the contrary, it enables him to hold his plugger more firmly.

No treatment has been successful, as it has been impossible to enforce rest.

#### DISCUSSION.

Dr. RIGGS, of St. Paul.—I have had a case similar to

the one described in a man who is suffering from writer's cramp in the right hand and right arm being affected, but it does not seem to have any special relation to the contraction of the trapezius muscle, the muscle affected in this instance. The reading of a newspaper seems to annoy him more than anything else, and when he endeavors to take up a newspaper, or look at anything very steadily, there will begin to develop clonic spasms of the trapezius muscle, most annoying and persistent. I have tried the usual remedies, and have got the most benefit at present from hydrobromate of hyoscine, but the case has been very discouraging, and hence the results obtained by Dr. Putnam have been quite pleasing to me.

Dr. STARR, of New York.—I can add to the statistics by saying I had a case of writer's cramp, in which the spasm extended to the muscles of the neck, and in which the patient's head was turned around in a decidedly typical position of torticollis, the face looking away from the arm that was affected, being turned around in this sort of way to the left; and I could not explain that very well, because, if we consider writer's cramp as due to fatigue of the motor cells of the right hand, then this man's fatigue was in the left hemisphere of the brain, but the turning of the head to the left would involve the motor area of the right brain, so that it did not seem that any spreading outward of motor impulses in one hemisphere would explain the position of the head at all. The man was a very neurotic individual, and I thought there was an element of hysteria about it, and found that he had, as many of these patients with cramp have, a point upon the surface of the body pressure of which would immediately stop the cramp. He had discovered this himself, or rather investigated at my suggestion, and found that pressure on the exit of the great occipital nerve would immediately arrest the cramp of the head, so that Dr. Shaefer rigged an apparatus by which the man could produce by means of a lever pressure on this particular point by simply putting the hand down to the side, so that he could correct the position. He wore that apparatus three years, and finally got well.

Dr. LESZYNSKY, of New York.—To what do you attribute the relief—hypnotism or atropine?

Dr. PUTNAM, of Buffalo. I attributed the relief entirely to the hypnotism, for the reason that he was able to write in the office, but was not able to write when out of the office, showing that there was a certain psychical

element that was lost by going away, but I think the rest of two and a half years must have had a tremendous influence.

Dr. BAKER, of Utica.—Possibly light will be thrown upon this question, if we consider how we go to work, to write, or to do anything intentionally. It seems to me sometimes that we should look upon these occipetive neuroses as being a sort of intention-contraction—something purely psychical, without the pathological basis that the intention-tremor has. When the boy begins to learn to write, he not only uses his fingers or his arms, but also about half the muscles in his body; in other words, the intention to do a thing involves not only the muscles required for the doing, but also an associated contraction or tension of a vast area of muscular structure. In reference to the cases where there is associated movement of the head or shoulder to one side it would not make much difference what the direction or what the area of muscular structure involved, if we consider that habitually, whenever we do anything, we associate in the doing of it a certain number of unnecessary muscular tensions. I warrant each one of us has some peculiar tensions of muscles which eventually establish the habit, attitude, whereby, when the associations become fairly enough fixed, there would be actually a spasm or contraction in some one direction; and so in the cure of the disease it seems to me that anything which will conduce to the breaking up of these associations will constitute the true remedy; and rest, of course, is necessary for the breaking up of any habit, while the change which comes about through hypnotism, or atropine, or anything else must be looked upon as one which comes usually, perhaps always through the process of breaking up associations that date back, possibly, to the beginnings of our intentional life.

# THE ETIOLOGY OF OBSTETRICAL PARALYSIS.

By G. L. WALTON, M.D., OF BOSTON.

## ABSTRACT.

This subject has not received the study it merits. The seat of lesion is probably in the brachial plexus. Carter's view, that the separation of the head from the shoulder stretches the plexus at the junction of the fifth and sixth roots, otherwise most satisfactory, does not explain the immunity of the pectoralis major. Oppenheim's theory of pressure against the clavicle does not explain involvement of the supra-scapular nerve and immunity of the pectoralis major.

A most satisfactory combination of these theories, with certain modifications, would be to suppose that the plexus is already brought up against the clavicle by rotations of the head away from the affected side, and that at the same time the supra-scapular nerve is put on the stretch between the point of the emergence and the bony edge around which it passes to reach the infra-spinous fossa. The separation of the head from the shoulder, which follows rotation in the strait, (the shoulder being held at the brim of the pelvis), still further stretches the supra-scapular nerve and still further bruises the plexus against the clavicle.

Accurate observations regarding position are desirable. The cases in which the position is noted tend, up to this time, to favor this mechanism, the right arm being generally affected in O. L. A. and O. R. P., the left in O. R. A.

## DISCUSSION.

DR. STARR, of New York.—I should like to ask whether any one here has seen an adult suffering from the relics of that condition. I see a number of these cases every year, and I uniformly give them a good prognosis, but it usually lasts several months, and the mother gets tired of coming, and I very rarely see the baby absolutely cured, and yet it seems to me, if they remain

we ought to see adults suffering from this condition, which I cannot say I have seen.

DR. TAYLOR, of Boston.—A few days ago I was told by a reliable observer, not a neurologist, that he had seen such a case at the Boston City Hospital.

DR. LESZYNSKY, of New York.—I have seen two cases in adults, one in a girl about twenty, and the other in a boy about seventeen. I do not know whether Dr. Walton refers particularly to cases without dislocation, or to cases where there has been traction made during birth, or whether to those cases which have occurred spontaneously. In one of these cases there had been a dislocation at the time of birth, and possibly this might be one of those post-traumatic cases where the joint has been affected. The prognosis was not always good. I saw one in a child fifteen months old not long ago, where there had been a dislocation; the dislocation was reduced, but so much damage had been done to the nerve-trunks that regeneration did not take place.

THE CHAIRMAN saw, this spring, a case in a child five or six years old that had dated from birth, the child coming to the hospital for some other affection.

DR. WALTON, of Boston.—I limited myself to pure and simple obstetrical paralysis, such as is sometimes produced in normal birth, without traction or the use of forceps, hook or finger, or anything of that sort. The prognosis of such paralysis would be different from that in which there is dislocation of the shoulder or any other local injury. I have not seen a case persist to adult life. I have seen, however, I think two cases, certainly one, at the hospital, in which an adult had fallen upon the shoulder and stretched the head apparently away from the shoulder and produced exactly this form of paralysis, affecting the upper arm group supplied through Erb's joint, and in such cases the supra-scapular nerve is also involved.

## DESCRIPTION OF BATHS, WITH EXHIBITION OF PHOTOGRAPHS AND DRAWINGS.

BY DR. RALPH L. PARSONS, OF SING SING.

### ABSTRACT.

The following description of a bath-room and baths, after plans furnished by Dr. Simon Baruch, and carried out by Mr. Frank Richter, is submitted to the Society as of interest, because of the completeness and efficiency of the baths, and the novelty of some of the arrangements.

The room, which is twelve by fourteen feet and a half in size, is situated in a basement, is well lighted and is adjacent to other rooms suitable for the ice-water tank, dressing room and heating apparatus, and is convenient to a water supply having a pressure of forty pounds to the inch.

At one corner of the room there is a hot air bath heated by hot water coils. This can be arranged for a vapor bath, or a moliere bath also, as desired. A thermometer, the stem of which is outside the bath, indicates the degree of heat. A hot water boiler, holding a hundred and fifty gallons, and heated by a hot water heater in the laundry adjacent, is situated in another corner of the room. The third corner is occupied by the rain bath, the needle bath, the bidet and the station for the Scotch douche. The remaining corner is occupied by a marble table, under which the pipes for securing the desired temperature and pressure are arranged, and upon which are placed the thermometer, pressure guage and also valves required for the baths, and also the pipes for the Scotch douche.

The floor of the room is of Portland cement, the ceiling is of iron and the walls are of brick, with the exception of the space back of the station for the Scotch douche, which is of marble. An ordinary bath tub is situated in the space between the hot water boiler and the needle bath. Valves for turning on the cold water are placed adjacent to the needle bath, so that a person



taking a needle bath by himself need not go to the table for the purpose of turning on the cold douche.

Diagrams and photographs illustrating the arrangement of the apparatus were exhibited.

In order to obtain the desired temperature and pressure for the needle bath the regulator is first opened; then the hot and the cold water valves are so turned as to produce the proper temperature; then the regulator is closed far enough to reduce the pressure to the desired force.

#### DISCUSSION.

Dr. ANGEL, of Rochester.—I am glad to see Dr. Parsons give his attention to the use of water in this way, and I was very much interested in the plan he has made to serve his purpose. I have had my attention somewhat attracted to the use of the cold douche with the force douche, such as Chareot and others have used in France with good effect, and I am always struck in many of these plans that there is no provision made for giving the large amount of water necessary. I believe they use a one-inch stream with a pressure of about thirty pounds graduated to circumstances. I do not know any one thing more valuable in the treatment of certain conditions, insomnia and neurasthenia, than the douche. I have suggested to Dr. Howard that it would be a very valuable plan in the routine treatment of many phases of mental troubles, and I believe our asylums for the insane eventually will be provided with many of the hydro-therapeutical measures.

Dr. TOMLINSON, of St. Peter.—I have a bath-room in the hospital I have charge of that I have always considered to be a model and especially efficient, because it makes provision for the elimination of places for the accumulation of dirt; in other words, the plumbing is so done that there is nothing in the room except the necessary stop-cocks to turn the water on and off. The floors and walls are tiled, making an absolutely plain surface which cannot get dirty, and in that room we are able to give every form of bath known to medicine although the room is only 14x24 feet.

Dr. PARSONS, of Sing Sing.—The pressure you have available is forty pounds, which is, of course, too much for most purposes.

Adjourned sine die at 1.05.

The following papers were read by title :

THE CEREBRAL FISSURES OF TWO PHILOSOPHERS, CHAUNCEY WRIGHT AND JAMES EDWARD OLIVER.

BY DR. BURT G. WILDER.

These men were recognized as superior in character and mental power. They were mathematicians and thought deeply upon the broadest questions. Wright was more of a writer and general critic; Oliver was more of a teacher of advanced mathematics. The latter was slight in frame and alert in action. The former was large of person and slow of speech and movement. Wright's brain weighed 1516 grams (53.50 oz.), Oliver's 1416 (49.94). Although above the average of male brains (about 1400—49.4) greater weights are not uncommon even among less intellectual persons. In both the frontal region is unusually high and wide; the unprecedented squareness of Wright's suggests some post mortem pressure, of which, however, there is no record. In both the supertemporal fissure is longer than common. Oliver's fissures present several individual variations of the common type, but none comparable with the two rare conditions in Wright's already noted by Dwight (*Amer. Acad. Arts and Sciences' Proceedings*, XIII., 210-215, 1877) and the writer (*JOURNAL NERVOUS AND MENTAL DISEASE*, XVII., 753-4; *Amer. Neurol. Trans.*, 1890; "Ref. Handbook Med. Sciences," VIII., 158-159, IX., 108). The complete interruption of the central fissure has been observed in a dozen or more cases. The simplicity of the fissures and the width and flatness of the gyres are paralleled in the Cornell collection only in the much smaller brain of an unknown mulatto (No. 322, "Ref. Handbook," VIII., Figs. 4767). Some approach to this condition occurs in Ruloff, a murderer (No. 965), and perhaps in a brain shown by Wagner ("Vorstudien" Taf. VI., Fig. 2) after Huschke ("Schädel, Hirn und Seele," Taf. V., Fig. 2). If fissural simplicity and gyral width

and flatness are family characteristics, or correlated with Wright's mental and physical deliberateness, then light may be thrown upon the problem by the conditions to be observed in his blood relations, or in others "slow but sure" in thought, speech and act. Since a close mate for the brain of Chauncey Wright has not been found in that of James Edward Oliver, the contemplated full account of it need not longer await the death of other moral and intellectual compeers. Such exceptional cases will always command attention. But all estimates of the extent and significance of their peculiarities will be only provisional until the careful comparison of many average brains supplies one or more types or standards. This necessity should be kept in the public mind.

# IRRIGATION OF THE POSTERIOR CEREBRAL FOSSA FOR THE RELIEF OF BASILAR MENINGITIS.<sup>1</sup>

By J. T. ESKRIDGE, M.D.,

Denver, Col.

Professor of Nervous and Mental Diseases and Medical Jurisprudence in the Medical Department of the University of Colorado; Neurologist to the Arapahoe County and St. Luke's Hospitals.

I AM not aware that trephining into the posterior cerebral fossa and cleaning it by irrigation have ever been practiced or suggested for the relief of basilar meningitis, except in Macewen's cases, in which the meningitis was local and occurred as a result of infection from disease of the middle ear.<sup>2</sup> Several have endeavored to relieve cerebral pressure by tapping the spinal canal at the upper cervical region and drawing off the accumulated fluid. Not long since a case of basilar meningitis limited to the posterior fossa, following an attack of pneumonia, came under my care, and the diseased process was so limited, that it occurred to me, that the pressure probably might have been relieved, and most of the results of the inflammation gotten rid of, by draining the cerebral fossa and washing it thoroughly through an opening made into the fossa through the occipital bone on each side of the median line. This case in detail is as follows:

Leonard M., male, aged 20, single, white, American by birth, employed as a dry-goods clerk, with a negative family history, had enjoyed good health in childhood, and his habits were said to have been excellent. He had never had a discharge from either ear, but on get-

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<sup>1</sup> Read by title.

<sup>2</sup> Dr. J. B. Roberts, of Philadelphia, trephined the skull for the relief of pressure in a case of tubercular meningitis, placing the trephine three-quarters of an inch above and three-quarters of an inch in front of the auricle. *Proceedings of the Philadelphia County Medical Society*, p. 465, for 1894.

In the *Times and Register* for 1894, Dr. B. Merrill Pickets published an article which I have not seen, entitled, "The Removal by Trephining of Fluid as the Result of Acute Cerebral Meningitis, with Report of a Case."

ting water into his ears, especially the right, on several occasions while in bathing, he had complained of pain in the latter ear. About March 18, 1895, he had an attack of pneumonia involving the base of the right lung. It was ushered in by a chill and followed by fever. The physician in attendance says, there was but slight consolidation of the lung, and the patient did not seem very ill, but made a rather tedious convalescence. He was able to return to his work about two weeks from the time of the beginning of his illness and continued on duty in the dry-goods store, until April 8, feeling rather weak, but complaining of no special ailment. On this date he worked all day, but in the evening began to complain of headache. By 10 P.M. the pain was excruciating, and localized in the front and back of the head, one side of the head not seeming to be involved more than the other. The next day, April 9, he had one or two chills, but the physician says he found no rise in temperature, but on the contrary, the axillary temperature was invariably sub-normal. He slept a great deal that day and seemed rather stupid; the following night he was restless. On Wednesday morning, the 10th, he was in much the same condition that he had been in the day before, although he did not complain of as much headache. During the afternoon he laughed and talked with his physician, and said he did not feel a particle of pain in his head. Early Thursday morning a slight difficulty in speech was observed, which seemed to be rather a difficulty in articulation, but with an effort he could make himself understood. He was irritable and restless, and it seemed to be difficult for him to concentrate his attention and keep it on any object long at a time. The temperature was normal. He was complaining then of pain in the back of the head. During the latter part of the forenoon he was delirious and restless, so that he became more or less unmanageable. At noon the temperature was slightly sub-normal; pulse 60; respiration 24. At 5.30 P.M. I saw him in consultation with Dr. Richmond, his attending physician. When I went into the room he was lying in bed, apparently asleep, but occasionally would open his eyes, stare, rub his forehead and then bury his head in the pillows. He was restless, throwing himself from one side of the bed to the other. He was considerably emaciated and depressed, corrugation of the forehead was observed, and the facial expression was that of suffering. His

mother reported that he had been yawning a great deal during the afternoon. On arousing him and asking him if he had headache, he replied emphatically, "No." On repeating the question he became irritable, and with great effort answered "No" quite loudly. I found it difficult to get him to concentrate his mind, and it was impossible for him to hold his attention on any one subject for more than a few seconds. The muscular tone and strength for one in his depressed condition appeared normal in legs and arms, but slight paresis of the left side of the face was apparent. The left pupil was a little dilated, and responded more slowly and feebly to light than the right. It was impossible on account of his mental condition, to examine the special senses or general sensory phenomena. The ophthalmoscope revealed a hyperemia of the optic discs. The physician informed me that he had taken his temperature but a few minutes before I saw the patient and found it  $98.3^{\circ}$ ; pulse, 60; respiration, 16. The deep reflexes were slightly lessened and the superficial absent. On account of the sub-normal temperature, which I cannot verify as I did not take it at that time, the dilatation and slow response of the left pupil, and the slight paresis of the left side of the face, an abscess of the brain was suspected, and the patient was removed to St. Luke's Hospital at my request. Two hours later on taking the temperature, it was found to be  $102^{\circ}$  in each axilla; the pulse was still slow, about 60, and respiration 28. The elevated temperature was suggestive of meningitis rather than abscess. One hour after my examination, at 5.30 P. M., he became totally unconscious and ceased to speak. He was quite restless and rubbed his forehead with his right hand considerably. On the next day Cheyne-Stokes respiration became apparent, lasted for an hour, and then was replaced by normal breathing. The posterior cervical muscles were observed to be slightly rigid, the knee-jerks were a little increased, and the apparent paresis of the left side of the face had almost entirely passed away. The pupils were equal and responded readily to light. From 8 A.M. of April 12, to 7 P.M., the temperature in each axilla ranged from  $102.4^{\circ}$  to  $103^{\circ}$ , and was found the same on each side of the body; the pulse varied from 70 to 76 and at times was intermittent; respiration was 36. During the early evening of April 12, the temperature descended about two degrees, pulse increased to  $78^{\circ}$ , and unconscious-

ness became profound. On the morning of the 13th the temperature was  $102^{\circ}$  in each axilla; pulse 92; respiration 34. He raised the left arm a number of times, but did not move the fingers of this hand. The left hand had been partially closed all night. During the next day, April 13, he remained in an unconscious condition, bowels were obstinately constipated, temperature varied from  $102.4^{\circ}$  to  $103.1^{\circ}$ , and was the same in each axilla; pulse was 92, respiration varied from 36 to 44. Retraction of the head became quite marked on the 14th; stupor gradually deepened, temperature remained about the same; pulse reached 118; respiration 38. At 5 A.M., of the 15th, the temperature was  $104^{\circ}$  in each axilla; pulse 180; respiration 56. At 8.10 A.M. the temperature was  $107.2^{\circ}$  in the rectum. He died ten minutes later. On the 11th and 12th the surface temperature on each side of the head was registered and found to be from  $2^{\circ}$  to  $2.5^{\circ}$  less than the axillary.

Autopsy, three hours after death, by E. R. Axtell, pathologist to the hospital. The brain only was allowed to be examined. The bones of the skull were thin. The dura and bones everywhere presented a normal appearance. The pacchionian bodies were prominent and the portion of the brain covered by them seemed to be slightly inflamed, but no pus was found here. The pia on the convex surface of the brain and over the base of the anterior and middle fossae presented a normal appearance. The parts lying in the posterior fossa were found covered with a thick layer of yellowish pus and inflammatory exudate and abundant watery fluid. The inflammatory material extended around the medulla. Both lateral ventricles were distended with an opaque watery fluid containing a small quantity of flocculent material. Considerable of this material was found in the fourth ventricle. The interior of the brain presented a normal appearance. A specimen of the lymph and pus was given Dr. Crouch, who reported as follows:—

“Cover glass preparation from the purulent lymph in the sub-arachnoid spaces showed the presence of *diplococcus lanceolatus* (pneumococcus) in great numbers and in pure cultures. Stroke cultures on glycerine agar resulted in almost pure cultures of the pneumococcus.

“H. C. CROUCH.”

This patient was removed from his home to the hospital because at the time of my first visit an abscess of

the brain was suspected, and if this had proven correct, an operation would have been necessary, but upon having the temperature carefully observed, and finding it elevated from four to five degrees, and especially when rigidity of the posterior cervical muscles developed, it was evident that I had a meningitis and not an abscess of the brain to deal with. While witnessing the post-mortem examination of the case, and seeing that the inflammatory products lay entirely in the posterior fossa, it occurred to me that in a similar case this condition might possibly be relieved by trephining into the fossa on each side of the median line and washing out the inflammatory products.

Three days later, or on April 18, I was asked by Dr. Richmond to see another case with him, at about 5 P. M. The history is as follows:—

J. N., aged 30, white, a boilermaker by occupation, whose family and personal history were not obtainable, suffered from La Grippe about four weeks before. He was greatly reduced by the attack, but no localized lesion was found. He had gradually recovered, so that he anticipated returning to work in a day or two, but he was still weak and languid. On the morning of the seventeenth, about thirty-six hours before I saw him, he awoke and was talking with his wife, when suddenly he put his right hand to his head and cried, "Oh my head!" Soon after this he lost consciousness, but rallied in the course of a few hours and talked some during the day, but seemed dull and stupid, and complained of a pain in his head. The next morning he was nearly unconscious, but in the afternoon he was able to talk with his wife and seemed rather bright. The temperature, Dr. Richmond reported, had appeared normal, although he had not registered it. When I entered the room at 5 P. M., he observed and spoke to me, and said that his head was paining him. He looked depressed and appeared to be suffering. Pupils were small, but responded to light. The posterior cervical muscles were rigid and the head slightly retracted. After I had been in the room examining him for two or three minutes, he became unconscious, and I found it impossible to arouse him. Temperature was  $98.3^{\circ}$  in each axilla; pulse 72; respiration 16. The knee-jerks were slightly increased. The ophthalmoscope showed no ocular change and there was no evidence of paresis or paralysis of any of the muscles. The history of La Grippe, the prolonged con-



valescence following it, and the sudden development of head pains with normal temperature, made me apprehensive of cerebral abscess, although no discharge was observed from either ear, and his wife stated that he had had no ear trouble. On removing him to the county hospital two hours later, his temperature was found to be 100.4° in the right axilla, 100.2° in the left, and 100.4° in the rectum. The rise in temperature was thought to be due to the disturbance caused by removing him to the hospital. He did not seem to be totally unconscious, his eyes would follow me or the lamp around the room, and when asked his name he endeavored to give it, and succeeded in doing so in an indistinct manner. I asked Dr. Rogers, the attending surgeon at the hospital, to see him with me at 9 P.M. The temperature then registered 99.6° in the right axilla, and 99.2° in the left; pulse 60; respiration 16. Retraction of the head was well marked, and on my attempting to bring the head forward, it seemed to give him some pain. The diagnosis at this time seemed to rest between meningitis and cerebral abscess. The retracted condition of the head indicated irritation of the meninges in the posterior cerebral fossa, but the normal temperature at the time of my first visit, and its again descending after he had been resting a few hours in the hospital, seemed to militate against the presence of meningitis. That night his temperature was registered in each axilla every two hours, and the average temperature for the night was 98.4°, being about the same in each axilla. At 9 A.M. the next morning the temperature in each axilla was 99.0°; pulse 64; respiration 18. He was becoming more stupid, pulse was occasionally irregular and respiration at times intermittent. About noon, when Dr. Rogers again saw him with me, his condition remained about the same as it had been during the morning, but we could find no indications for operative procedure. During the afternoon his temperature varied from 98.6° to 99.4°; pulse 63; respiration 18. It was quite evident that there was pressure in the posterior fossa, especially around the pons and medulla. The low temperature contra-indicated meningitis, although normal or sub-normal temperature is sometimes observed in this disease. Abscess of the brain did not seem likely as the temperature had been found constantly a little above normal for the twenty-four hours he had remained in the hospital. During the afternoon he seemed to be sink-

into a deep comatose condition, and it was evident that life could not be prolonged many hours unless relief of pressure of the pons and medulla were effected by surgical means. I could be positive of but one thing, and that was pressure at the base of the brain in the posterior fossa, and I asked Dr. Rogers if he would trephine and endeavor to remove, by washing, any inflammatory material that we might find in the posterior fossa. To this he agreed, and the operation was set for 8.30 P.M. Just before the operation he was totally unconscious; temperature was  $99^{\circ}$  in the right axilla, and  $99.2^{\circ}$  in the left; pulse 64; respiration 18. Retraction of the head was well marked, and he presented the appearance of one dying from intra-cranial pressure. Although he appeared totally unconscious, yet it was found by pricking him with a pin, movements of the limbs would take place, so that we were compelled to use an anæsthetic. Just at the time of beginning with the chloroform the pulse was found to register 78; as soon as he became anæsthetized it fell to 54.

Description of the operation by Dr. Rogers.

The operation was begun about 9 P.M. A curved incision was made and the tissue stripped downward so as to expose the skull to as low a point as possible. A three-fourth inch trephine was then used, and the opening was slightly enlarged latterly with a rongeur forceps. It was subsequently shown that the opening was about on the level of the posterior margin of the foramen magnum, and about three-fourths of an inch to the left of the median line of the occipital bone. All tissues appeared normal, the veins being slightly engorged. The dura was then laid open transversely for about three fourths of an inch. A large amount of cerebrospinal fluid escaped. This could not be measured, but it seemed to be a greater quantity than would normally be present. The cerebellum could be easily raised from the dura, and when the flow had abated, a soft catheter was passed in through the opening in the dura, and the sub-dural spaces freely irrigated in all directions with normal salt solution. To the right of the median line the catheter easily passed without obstruction, a distance of over two inches. It was subsequently shown without much doubt that this passed across into the right fossa. The salt solution returned freely. The pulse at the beginning of the operation raised to 140, but fell during the irrigation to 112, and the respiration became deeper and more regular.

A piece of gauze was left as a temporary drainage for what fluid might be left, so as to slightly separate the edges of the dura. With this exception the wound was completely closed.

This drainage was removed the next day, and the wound allowed to close completely. No great difficulty or serious symptoms occurred during any part of the operation.

Further remarks by Dr. Eskridge.

Immediately after the operation the temperature in the right axilla was  $99.6^{\circ}$ , and in the left  $100.5^{\circ}$ ; pulse 114; respiration 24. At 2.30 o'clock the next morning the temperature registered in the right axilla  $100.6^{\circ}$ , left  $100.8^{\circ}$ . The pulse was then 120, respiration 24. Two hours later the temperature had risen to  $102^{\circ}$  in each axilla, pulse and temperature remaining the same as before. At 8.30 A.M. the temperature had fallen to  $101^{\circ}$ , where it remained the greater portion of the forenoon. A few hours after the operation the patient opened his eyes and looked around the room. During the forenoon succeeding the operation the patient presented an improved appearance. He would open his eyes and try to protrude his tongue when requested to do so, but was unable to get it beyond the teeth. In the afternoon, when visited by his wife, he seemed to recognize her and succeeded in protruding the tongue at the request of the nurse. He was able at this time to empty the bladder, which he had not done the previous forty eight hours. He kept his eyes open for about three-quarters of an hour and took notice of what was going on around him. On the morning of the second day the temperature ascended to  $103^{\circ}$ ; pulse 154; respiration 14. He was then comatose, rapidly failed and died just before midnight.

Autopsy eleven hours after death by Dr. Leonard Freeman, pathologist to the hospital. The wound had healed without the formation of any pus, no abnormal adhesions between the dura and the bone were detected. The external surface of the dura presented a healthy and glistening appearance. The pia over the convex surface exhibited no marked changes except engorgement of the veins. No clots were found in the venous sinuses. The arteries of the base of the brain presented evidences of disease in their walls, and in the main branch of the left middle cerebral artery, just before the cortical vessels are given off, a clot was found which

blocked up the entire calibre of the vessel. In the centrum ovale of the left frontal lobe a considerable quantity of semi-fluid blood was found, which after plowing up and destroying a considerable portion of this part of the brain, ruptured into the lateral ventricle, and filled the lateral, third and fourth ventricles. The corpora striata were softened. A slight hæmorrhagic extravasation was found in the right frontal lobe. The remainder of the brain presented a normal appearance. There was no evidence of any meningitis nor of abscess. The pathological condition was one of hæmorrhage into both frontal lobes, the greater in the right, the blood finding its way into the lateral ventricles, and filling these together with the third and fourth ventricles.

In the clinical history and pathology of this case there are many points that invite discussion, but the main subject to which I wish to call attention in this communication is the probable effect of draining and irrigating the posterior cerebral fossæ in basilar meningitis. Unfortunately the absence of meningitis in this case prevented us from being able to determine practically the results likely to follow the surgical measures resorted to for the relief of this trouble. The brilliant results obtained by Macewen from irrigating one-half of the posterior cerebral fossa in localized meningitis encouraged me to hope that a more desperate condition might be relieved by more elaborate, but somewhat similar surgical procedure. I had first planned to have Dr. Rogers enter the posterior cerebral fossa by two trephine openings, one on each side of the median line, but on his exposing the left side, finding no pus and being able to pass a soft catheter over to the other side and irrigate it, it seemed that the removal of another button of bone was unnecessary. In a case of meningitis practical experience will teach whether the removal of one button of bone will suffice to enable the surgeon to accomplish all that he can hope to do from draining and irrigating the basilar surface of the posterior portion of the brain. It is probable that in general basilar meningitis the measures resorted to in this case would do nothing more than slightly prolong life by relieving pressure on the parts in the posterior fossa, but that in numerous cases of cerebro-spinal meningitis in which the cerebral mischief is almost entirely limited to the posterior fossa, they may result in the cure of the disease. The result of the operation in the case here

reported seems to demonstrate that the posterior cerebral fossa may be drained and irrigated during life by means of a trephine opening into the fossa, on one side, and in properly selected cases, may give relief to the patient. It was evident after the operation that the patient's respiratory and cardiac centres acted much better than before it. The man's improved general condition and his partial restoration to consciousness for a period of twenty-four hours after the operation indicated that general intra-cranial pressure had been partially relieved by the surgical measures resorted to.

I wish to thank my colleague, Dr. Rogers, for his co-operation in this case, for the skillful manner in which he performed the operation, and for his courtesy in giving me a description of it for this paper.<sup>3</sup>

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<sup>3</sup> Microscopic examination of the diseased cerebral vessels show them to be affected with syphilis.

## THE HOME TREATMENT OF INSANITY.

By H. M. BANNISTER, M.D.,

Chicago

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IN his address before the American Medico-Psychological Association, in 1894, Dr. Weir Mitchell, one of the first of America's specialists in neurology used the following words: "You hold to and teach certain opinions which we have long learned to lose. One is the superstition (almost is at that) to the effect that an asylum is in itself curative. You hear the regret in every report that patients are not sent soon enough, as if you had ways of curing which we have not. Upon my word, I think asylum life is deadly to the insane. Poverty, risk, fear send to you of true need many patients; many more are sent by people quite able to have their friends treated outside. They are placed in asylums because of the widespread belief, you have so long and, as we think, unreasonably fostered to the effect that there is some mysterious therapeutic influence to be found behind your walls and locked doors. We hold the reverse opinion, and think your hospitals are never to be used save as the last resource."

In a paper on the Provisional Treatment of Insanity, read before the Illinois State Medical Society, in 1894, Dr. Sanger Brown, another leading neurologist, states: "It should be remembered at this point that every insane patient could be treated more effectively out of an asylum than in one; that is, this proposition is theoretically true. If the services of a practical alienist are available, it is always safe to say, providing the question of expense may be left out, that any and every case of insanity can be best treated outside of an asylum. That is, if it is an acute attack in a young person and the prognosis favorable, considerable pecuniary sacrifice ought to be made for some months in order to furnish proper treatment outside of an asylum."

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<sup>1</sup> Read by title.

The author of a recent excellent manual of nervous diseases states incidentally to the treatment of melancholia, that he is no believer in asylum treatment for curable cases of insanity, a statement that is as explicit and unqualified, though not so specially emphasized and amplified, as those before quoted.

When such views as these are held by high authorities, some notice of them is perfectly justifiable. If they are wrong, they are dangerous not only to individuals, but to the public; if right, then all our institutions for the insane are on the wrong track, they are simply asylums for the detention of an inconvenient class of afflicted incurables, and the term hospital is a misnomer. I need not speak of their inconsistency with the plea, in the address referred to, for the reform of these institutions and the improvement of their hospital functions. If asylum life is deadly to the insane, if as hospitals they are "only to be used as a last resource," if "every insane patient could be treated more effectively out of an asylum than in one," and they are no places for cases that are still curable, we might as well confine our efforts to making them simply places of humane detention and restraint for the later stages of mental decay without therapeutic interest, and only affording scientific results in their generally brief ante-mortem records and the possible findings of autopsies. That this is an ideal to be looked forward to would not be admitted by any of those I have quoted, but it is the legitimate deduction from their utterances which I have verbally quoted, and anything they have stated to the contrary is, at best, grossly inconsistent with these deliberate statements. Dr. Mitchell's ideal hospital would be a needless extravagance if insanity could be as well treated outside of it, for the insane are mostly public charges, maintained by the taxpayers who have no wish to have more done at their expense than necessity and simple humanity demands.

The questions here to be considered are two: first, can insanity be as well treated outside of an asylum as within its walls? second, has the hospital or asylum, by itself any direct curative influence in cases of insanity? These questions are somewhat interdependent, but neither includes the other, and I shall, therefore, consider them separately.

First, can insanity be treated as successfully outside of an asylum or hospital for the insane as it can within

such an institution? The claim as stated is that it can be better so treated, but if the negative is proven to the question as here stated, that is all that is needful. It is difficult to define insanity, and I would wish to use the term in the sense that will be accepted as perfectly fair to both sides of the argument, and to which no exceptions can be taken. It ought, however, to include only the so-called curable forms, for these are the ones in dispute; those in which hospital or asylum care is the "last resource," may reasonably be left unconsidered. Confining the term, therefore, to acute or curable cases, we may understand by it the ordinary cases of mania and melancholia, or acute, excited or depressed insanity, which form almost the whole of what may be called the curable cases. Indeed, with the exception of a few cases of toxic delirium and a few of a certain type of primary dementia they form the whole series. Paranoia in delusional insanity may be excluded as a degenerative type and rarely curable, and when it does not appear in its advent under one of these forms its beginnings are insidious and generally overlooked. Paresis and all the organic insanities may also be left out of consideration here, though there is generally little question as to whether extra asylum treatment is preferable in any particular case.

When we consider that the great mass of the insane come from the poorer classes, it will be readily seen that the exceptions made by Dr. Sanger Brown form the rule, the question of expense alone will decide the impracticability of extra asylum treatment. This is especially true in maniacal cases which, it is readily seen, cannot be properly managed in an ordinary home without incurring expenses far beyond the means of the average citizen. The conditions in an average country home are well illustrated by the brutal reply I once heard given to a relative in a hospital who was subject to occasional spells of excitement and who asked to be taken home. The answer was that if he came, they would have to prepare a cage for him in the back premises away from the family and house. It is in such cases of home treatment that the greatest abuses of the insane occurs, abuses that ought not to happen even in the worst regulated asylums. I have had ample personal testimony as to this fact, both from the active and the passive participants. When actual abuse or hardship to the insane is lacking, there are yet necessarily employed measures



that are not desirable or helpful. Mechanical restraint is one of these, it can be dispensed with in a well-managed hospital for the insane and save in altogether exceptional,—mostly surgical,—cases it is rarely useful or expedient. But in caring for a violent maniac at his home, where financial conditions are not such as will permit the employment of one or more skilled attendants, even were such to be had, it is almost or altogether indispensable. The only possible alternative is absolute seclusion in a strong room, which must be improvised roughly and very imperfectly, or the rough manual restraint of ignorant and often brutal hirelings or stupefaction with narcotics by the attending physician. The effect of this home treatment, according to my own observation as an asylum physician, receiving its results under my care, is too often incurable insanity, and what Dr. Weir Mitchell calls a superstition on the part of alienists, has such experience for a rational basis and origin.

If, however, the patient's means are such as to make expense a matter of small importance, or if even at considerable sacrifices all possible provisions and appliances can be had, I doubt whether even the home treatment is the best for the patient in acute mania. In the first place, proper attendants are hard to get. In a large hospital there are always numbers to select from, and a reasonably competent observer and judge of human nature can man his wards for acute cases with fairly competent and reliable help. It ought to be remembered, moreover, that a good attendant on the insane cannot be made by training alone; something is required of natural fitness for the position. Training as a nurse does not properly qualify anyone for such a work; moreover, the available trained nurses are almost exclusively females, and on this account alone, unfit to care for masculine maniacs, and they would have to be very exceptional ones to be willing to undertake such task. In the second place, there is a disadvantage to the insane patient in retaining him in accustomed surroundings. The glimmerings of reason that remain in the general excited state are apt, in such cases, to have in themselves a deleterious effect, if I may so express it. If he recognizes his surroundings and resents the necessary restraints put upon him, it is far better to have him separated from his family, so that he may not in his unreason acquire perversion of feeling toward them. This may not seem to be,

at first sight, a serious danger, but it is one I should myself wish to sedulously guard against.

Lastly, even an ordinarily well equipped and well managed asylum has far better accommodations for the maniac than the best appointed home can have. Apart from the sentimental objection, of which I realize the strength, it is infinitely preferable. If all asylums were what they ought to be, and what I believe many of them are, scientifically conducted and under thoroughly competent alienists, this objection ought never to exist. Of all forms of insanity acute mania is the most amenable to hospital treatment, and the one that furnishes the largest proportion of recoveries in our institutions for the insane. I cannot say as much for it under home treatment.

With the depressive forms of insanity the case is somewhat different, many, perhaps a large majority, of the milder cases recover without being sent to any institution. Many, indeed, are walking cases, either undergoing no treatment at all, or are office patients of neurological specialists, and these, I believe, are to some extent responsible for the views I am here combatting. Yet these are just the cases that furnish the tragedies that we read of daily, the suicides, and no small proportion of the homicides. There is no question but that prompt asylum care would prevent many of these, and in that point of view the question is easily decided: It is impracticable, however, to have such cases committed, as a rule, and, therefore, extra asylum treatment only is available. In cases of pronounced melancholic frenzy, especially with suicidal tendencies, the asylum is the only and not the last resource of the poor, and furnishes decided advantages of safety to those who are better off. Next to mania, melancholia furnishes the largest proportion of hospital cures, while its treatment at home furnishes a very much larger proportion of asylum incurables.

The great need for the milder melancholic cases is cheap sanitariums where, like ordinary neurasthenic patients, they can go voluntarily and be under medical care and observation, having all the advantages of a hospital for the insane without the name. Such institutions, where they now exist, are far too expensive for the great mass of these patients, and the lack of inexpensive establishments costs the country annually many lives and inflicts untold misery to families, relatives and friends. The severer forms are best treated in the asy-

lums, where they can be under complete control. In neither class, as a rule, is treatment at their homes advisable or safe, in my opinion.

The only forms of acute insanity that can advisably be treated at ordinary homes (meaning by "advisably" that under circumstances, or existing conditions, it may seem best) are certain types of primary dementia and certain delirious forms from exhausting disease, with great physical prostration. If, in these last, mental improvement does not take place with that of the bodily health, sequestration and change of scene are likely to become necessary. Mild cases of chronic aberration can, of course, be cared for anywhere, but there are some objections on their own account, and on that of others, which, however, I need not dwell upon here, but will refer to later on.

One of the strongest arguments against the practicability of proper home treatment for acute insanity is found in the fact that general hospitals will not assume the care of insane cases. With all their appliances, their large consulting and resident staff, their numerous trained nurses, they find active insanity something out of their province, and inconvenient to handle. I have known of a number of instances where the insane of the less pronouncedly demonstrative class were temporarily detained in a general hospital, but I cannot recall any appreciable proportion of cures effected there, and in all cases the patients were finally compelled to leave. I have seen but one case of active maniacal excitement in a general hospital, he was detained only a couple of days and was not handled as would have been thought best in a well regulated asylum for the insane.

Thus far I have not referred to what seems to me the chief objection to the home treatment of the insane, in the comparatively few cases where it is not impracticable on other grounds. It is one also that seems to have been acquired by the writers that I have quoted, though as medical observers they must have been aware of its existence. I refer to the influence of the presence of the insane upon others, especially those nearly connected with them by blood and heredity. One insane member of a family often means a family tendency to insanity, and where there does not appear to be any actual insane taint there is, perhaps, a family constitution that may have a very unfortunate influence. The suggestion of a family liability is constantly presented by

the presence of the disease at the home, and often has disastrous effects. It would be far better that every such case should be as much as possible out of sight and out of mind. Since starting to write this paper I have been consulted by a gentleman who is threatened with melancholia, has it, indeed, in its earlier stages, the cause for which can only be found in the retention at home of an insane brother. There is, in fact, a well-recognized mental contagion of derangement which constitutes a serious danger, especially to those who are in the least degree bearers of a hereditary taint, or those who are liable to be seriously emotionally disturbed by contact or association with the insane. The effect on the young children of a family is also one that is to be seriously considered, they are particularly susceptible to injury from this cause, and it may be an injury to them for all their future lives.

The second question is, does the hospital have in itself any curative effect on the disorder as I maintained be answered positively in the affirmative. Its therapeutic influence is, moreover, very far from being mysterious, it is based on well-known principles that should suggest themselves to any one who gives the least attention to the matter. In the first place it is a change of scene, a removal from the habitual associations and those under which the mental disorder has developed and increased. This alone is a very important matter and one that has often a directly curative effect. Even old cases of insanity, regarded as incurable, are often amenable to this influence. I have seen several cases of recovery from chronic and apparently hopeless insanity follow directly the transfer to a different hospital. With acute cases this effect is often very marked, recovery follows the change without any other apparent reason. It affords to some a beneficial shock, a mental stimulus, and in this way it may be especially useful in certain hysterical and hypochondriacal cases. I can recall an instance of an hysterically "paralyzed" patient who had for thirteen years gone the rounds of practitioners and specialists, and who made a good recovery, was out and earning his living all within six months after his committal as insane to the hospital. While I had the treatment of this patient myself, and, I believe, used all due therapeutic diligence to favor his recovery, I must admit that I consider that the hospital influence had the most to do with it.

The regulated discipline of the asylum has a very beneficial effect on most acute cases of insanity. It is the regular thing in a well-conducted asylum to see a man, admitted a raving maniac often brought manacled or strapped, sitting quietly in the ward within a few minutes after his reception. This is the more noticeable as it is not accounted for as a rule by anything other than the general atmosphere of the place; it is the immediate effect of the change and the surroundings. In some cases this is not observed, and in most maniacal cases there are some subsequent outbreaks, but it is so common as to be the rule. This influence is not temporary, as a rule it continues to a greater or less degree. If I am called on to account for it, I should have to say that I believe it the complex effect of change, removal of irritation, and the unconscious recognition of a kindly but firm authority, all of which are conditions best provided in a well-regulated hospital for the insane. It is a "mind cure" in a double sense. If I am not greatly mistaken, some of the benefit of the well known "rest-cure" treatment, devised and so successfully carried out by Weir Mitchell in certain forms of neurasthenia, is due to the same causes. As I have said elsewhere, in these days when so much is said as to suggestion in therapeutics, the hospital is the strongest of all therapeutic suggestions.

The mere removal from domestic irritations and responsibilities has in itself often a most beneficial effect. Every large insane hospital, I believe, contains patients who show no symptoms whatever of aberration during their hospital residence, but who are unable to endure even the shortest sojourns at their homes without a mental breakdown. Sometimes this appears in dangerous forms, as for example, in an old lady who was for over twenty years an inmate of several western asylums and at all times amenable and apparently perfectly rational. Repeated trials at home, however, always developed within a very short time the most pronounced homicidal insanity which, on two occasions, had had very serious consequences. I might name other cases of my own observation in which hospital residence was the only salvation from active insanity, but it is unnecessary. In recent cases the home responsibilities and worries, the sense of mastership that exists at the patient's residence, have all a bad effect which is relieved by transfer to the hospital, and in this way the change is

sometimes directly curative. It is hardly necessary to say here that for the great majority of those whom it receives, the poorest State hospital, worthy of the name, offers advantages in the way of architectural arrangements, administration of food and medicines, baths, attendance and oversight, over what they could possibly receive at home.

The only conditions I can conceive of under which an asylum could be "deadly to the insane" are first in certain convalescing cases when the change from hospital to home life is essential to complete the cure, and cannot on account of lack of friends be had, and possibly in some few exceptional patients that from some idiosyncrasy of mental make-up are unsuited for asylum treatment, or who, still retaining some mentality, from a marked fear or prejudice against hospitals, would have their condition aggravated by such a change. The first class are not so rare, there comes a time when with certain patients the hospital can do no more good and then removal is advisable. When this cannot be done, and they are not capable of caring for themselves, they may suffer, and such cases form one of the most troublesome problems for the asylum physician. The sufferers from an anti-asylum idiosyncrasy must be few as I cannot recall a case, but the sufferers from anti-asylum prejudice are numerous enough, though it rarely has any effect other than that of perhaps strengthening the moral impression made by the change to the hospital and, therefore, possibly of sometimes indirectly aiding the cure. I can imagine, however, that there may be individuals in whom it might be so intense as to cause injurious effects. The principal evil it does is to delay all active measures to make the hospital the last resource for those who can afford no other effective treatment as well as for the small minority who can. Poverty, fear, and risk, send "of true need" the vast majority of the inmates of public asylums, and it is a great misfortune that the poverty should be overtaxed, the fear and risk augmented by prejudice largely based on a miserable political system that has too much prevailed in our asylums and unduly supported by the ill-considered utterances of those who ought to know better and who would hardly be ready to attempt a serious defense of their statements.

I believe that I have said enough to prove that insanity in the vast majority of cases can be best treated

in a special institution or hospital for the insane, and that the hospital itself has a certain curative influence. But in this connection I cannot do better than quote from one who ranks second to none as a neurologist, and who was at the time what I quote was written one of the severest critics of the existing deficiencies of American asylums, Dr. E. C. Spitzka.

"An asylum sojourn has in the vast majority of cases a good effect on the insane. Curable patients are never injured in their prospects as to curability in a medically well-managed institution, and incurable patients should be there for practical reasons, and are usually better off in than out of an asylum.

"The advantage of asylum treatment are the following: 1. Refusal of food and medicines, the great obstacles to the treatment of the insane outside of asylum, are best dealt with by a skillful corps of physicians and attendants always on the spot, with the necessary appliances at their disposal. 2. The necessary supervision of the insane at all hours can be carried on with the least expense and greatest thoroughness in the asylum ward. 3. The excessive and damaging use of narcotics, calmatives, and restraint, necessary for the purpose of preventing scandal in the neighborhood, and noise, destructiveness, and exhaustion at home, can be dispensed with in the asylum. 4. The sojourn of a patient in an asylum, the continual reminder which the restraint of its walls is to him that he is considered insane—whether he believes himself to be so or not—is in many cases a far stronger incentive to a kind of reflection which leads to the correction of delusions than any drug."

What Dr. Spitzka says is certainly true of a medically well-managed institution, such as I believe many of the hospitals for the insane are at the present time. Much of it will also be true even of one with many medical deficiencies like others now existing. But an institution will have to be very poor, indeed, and woefully mismanaged not to be a better place for the care of the insane than in the homes of many of these unfortunates.

I am not in this paper, I wish it to be understood, defending asylums against the criticisms that have been made, and which are often too well deserved. When appointments of medical staffs and chiefs are made, not

on merit, but for no other qualifications as far as known or can be ascertained, than political influence or services, as we know to be sometimes the case, it is impossible but that many of them should be very far from perfection. The misfortune of this is the greater in that it destroys the confidence the public ought to have in these institutions, and keeps up the damaging prejudice to which I have referred. It must be kept in mind, however, that a very moderately qualified physician, unfit to be called an alienist, ought to be able, with the assistance and appliances of an asylum, to do much better for the insane than a more skillful practitioner could possibly do in ordinary general practice. The mistakes he might make other than those of erroneous diagnosis of recovery and premature or too-delayed discharge (and these are serious enough) are, on the whole, less likely to be dangerous to the welfare of the patient and others, than the accidents that might occur outside. This is especially true, as I think will be seen by anyone on reflection, in cases of active mania or suicidal frenzied melancholia.

The remarks I have made thus far, apply mainly to public asylums or hospitals, since they are the first and only as well as the last resource of nine-tenths of the cases of acute insanity in our general population. Private asylums are comparatively few in number, and are available only for the minority to whom expense is a secondary consideration. They are not so much in this country, also the objects of distrust, as are the public institutions, which, owing to the element of politics in their control and management, and have in times past too largely merited the criticisms given them. But even in their worst days they have been a blessing to the unfortunate and have fulfilled a very necessary function in the State.



# THE ASSOCIATION OF TABES AND PARALYTIC DEMENTIA. REPORT OF FIVE CASES.<sup>1</sup>

BY THEODORE DILLER, M.D., PITTSBURG.

## ABSTRACT.

There is a history of syphilis in at least four-fifths of all cases of tabes, and two-thirds of all cases of paralytic dementia. The development of paresis in cases of tabes, and *vice versa*, has been noted by a number of observers. The distinction between true tabes and mere tabetiform symptoms so frequently noted in paresis must, as Mickle has pointed out, be sharply made. The theory of Fournier, Strumpel and others, that tabes and paresis arise from the same morbid process, affecting in the first case the cord and in the second the brain, has much to commend it, but cannot as yet be considered as established.

The association of tabes and paresis, however, is an important and interesting clinical fact, which has not been sufficiently noted in works on nervous diseases.

Five cases (all men) were cited in which paresis and tabes co-existed. The symptoms in all cases clearly pointed to this condition. In four of the cases the symptoms of paresis were engrafted upon those of tabes. In the fifth case this point could not be ascertained. In two cases there was a clear history of syphilis. It was not known whether any of the other three were syphilitic.

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<sup>1</sup> Paper read by title at the meeting of the American Neurological Association held in Boston, June 8th, 1865.

# A STUDY OF THREE CASES OF TUMOR OF THE BRAIN, IN WHICH OPERATION WAS PERFORMED — ONE RECOVERY, TWO DEATHS.<sup>1</sup>

BY LEO. STIEGLITZ, M.D., NEW YORK.

CASE I. Glio-sarcoma. The size of an almond; developing two years and ten months after the successful operation of a cystic tumor of the left motor area. Successful localization and excision. Recovery.

The symptoms in this case prior to the first operation in June, 1892, consisted of focal epileptic discharges in the right arm with occasional general convulsions. Three months after the onset of the focal epileptic discharges, paralysis began to develop in the muscles affected by the focal seizures. In spite of the absence of all general cerebral symptoms, notably headache and choked disc, the diagnosis was made of a tumor of the brain, involving part of the arm centre, and an operation was recommended. The operation was performed by Dr. Arpad G. Gerster in June, 1892, and revealed the presence of a small cystic tumor, which was evacuated and allowed to heal under drainage. The walls of the cyst were perfectly smooth, and no sign of any further growth was found. The patient made a good recovery. Excepting occasional focal discharges and three or four general convulsions, she remained perfectly well for the next two and a half years. In February, 1895, she complained for the first time of headache, which was quite severe and persisted for two weeks. At the same time the signs of a beginning papillitis were found, and the right arm, which had recovered a good part of its original power, showed again a marked loss of power. The diagnosis was made of a refilling of the old cyst or a recurrence of the new growth upon the basis of which the cyst had probably originally developed, and ele-

<sup>1</sup> Read by title at the Meeting of the American Neurological Association, held in Boston, June 8, 1895.

ments of which had in all probability been left in the wall of the cyst. Another operation being urged, Dr. Gerster entered the skull at the old aperture, which had been left covered by a gold plate. A small new growth, not larger than an almond, was exposed at the point where the old cyst had been. This new growth was carefully excised by Dr. Gerster, and then a circle of brain tissue which had surrounded it, and which was apparently partly infiltrated by the neoplasm was excised. The patient again made a good recovery, excepting three seizures which occurred immediately upon the removal of the original dressing. The patient has been well since the operation; the paralyzed arm is regaining its power slowly. The microscopic examination of the new growth revealed the presence of the old cyst wall, and starting from one point of it a *glio-sarcoma*; the surrounding brain tissue, which had been excised, was found partly infiltrated with elements of a *glio melano-fibro-sarcoma*.

The author draws the following conclusions from the case:

1. A new growth may be present in the brain without causing any general cerebral symptoms whatsoever; in order to make an early diagnosis of the presence of a new growth it is well, therefore, to be guided by the symptoms present rather than by the absence of other customary symptoms.

2. A cystic tumor of the brain not of parasitic origin is not as innocent in character as many would believe.

3. The simple evacuation of such a cyst when found, is not sufficient; if it is surgically possible, the wall and the surrounding brain tissue should be excised in order to prevent a future growth of the malignant elements left in the wall of the evacuated cyst.

4. If the immediate excision is not possible, it would be advisable to open the wound a few months later and attempt to excise the collapsed cyst wall, which is compressed into a small space by the general cerebral pressure in the same way in which an abscess cavity is quickly obliterated after evacuation.

CASE II. *Glio sarcoma* of the right cerebellar hemisphere—successful localization, operation, death, autopsy.

The symptoms offered by the patient during life were headache, choked discs, vomiting, spells of vertigo, occasional staggering to the right side, deafness in

the right ear, partial reaction of degeneration to the electric currents in the right side of the face, although no trace of paralysis was present till some months later, and hyperæsthesia to the galvanic current, with reversal of formula in the left ear. The diagnosis was made of a new growth of the cerebellum compressing the right seventh and eighth nerves, prior to, or at, their common entrance at the internal auditory meatus. The patient's condition became so miserable that it was agreed to make an effort to relieve him by surgical means. Dr. Gerster proposed entering the skull in the region of the right occipital lobe, raising the latter and entering the posterior cranial fossa by an opening through the tentorium. On October 27, 1893, the operation was performed; there was tremendous intracranial pressure, producing quickly a large untractable hernia cerebri; although Dr. Gerster succeeded in exposing the tentorium, the operation could not be completed on account of the great intra-cranial pressure. The patient rallied well from the operation itself; the wound was infected during a change of dressings, however, and the patient died of purulent meningitis. At the autopsy a gliosarcoma of the cerebellum, of the size of a large walnut, was found at the point to which the symptoms during life had pointed.

CASE III. Spindle-cell sarcoma of the left arm, face and speech centres—successful localization, operation, death, autopsy.

The symptoms presented were the occurrence of five or six focal epileptic seizures, involving the right side of the face and the right arm, slight loss of power in the right face and arm, motor aphasia, headache, mental hebetude, and a point in the left fronto-parietal region of the skull which was exquisitely sensitive to tapping. The diagnosis was made of a brain tumor, involving the face and probably the arm and speech centres in the lower half of the left central convolution; on account of the involvement of the motor speech centre rather forward than backward of the fissure of Rolando. After repeated delays the operation, which had been recommended as soon as specific treatment had proved futile, was performed by Dr. Howard Lilienthal. By the time the dura was well exposed the cachetic patient had lost so much blood, the diploe bleeding profusely, that it was decided to postpone opening the dura to a subsequent operation. The patient, however, never rallied from

the shock of the operation and the loss of blood, and died twenty-nine hours after the operation. The autopsy showed the presence of a sarcoma involving the arm, face and motor speech centres in the left motor area. the opening in the skull corresponded to the upper half of the tumor; had the dura been opened, the new growth would have been exposed; the growth was imbedded in the brain substance in such a way that it could have been enucleated and removed through an opening sufficiently large.

## Asylum Notes.

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By R. M. PHELPS, M.D.

Rochester, Minn.

### *The Boundary Lines of Insanity.*

"Insanity is a disease." So we iterate and reiterate almost daily to our nurses, to the profession, and to the public. And yet its definition into a disease almost eludes our grasp at times. A disease has limited duration usually. Insanity hardly has any. Disease usually runs to a climax, and proceeds to death or a receding. Insanity may, but often does not, or does it but vaguely. Disease, not rising to a climax, is usually progressive. Insanity often is not. Disease has lesions which we may quite definitely assign. Much of insanity has only a vague and theoretical basis,—much of it none even theoretical. Disease can not last a year in seeming intensity and then be gone in a breath. Insanity can. Disease cannot come and go in waves of months duration. Insanity often does.

In short, disease usually has fairly well defined boundary lines, insanity often has not. Not that we should marvel at this too much. Doubtless it is as mysterious that disease should rise to a climax and then recede, as that it should go on indefinitely. Indeed, much more so, than to be steadily progressive till death. For why should its cause cease acting? What is the susceptibility that is exhausted? What is the immunity that is acquired?

Insanity as a disease with prodromes, onset, climax, and a receding toward chronicity or to cure, must then be regarded as inextricably interlinked with both "defectiveness, congenital," with its occasional active or progressive periods (idiocy, imbecility and feeble-mindedness), and with "defectiveness inherited," as inherent in brain and nerves, vaguely definable, and by reason of which, without regard to apparent brightness or mental activity, the person's mental balance is easily over-

thrown. When we say "insanity is a disease" then we include varying degrees or forms of defectiveness.

And then for "boundary lines," where are they? Not only may we ask what divides acute delirium from acute mania, as did H. C. Wood (*Amer. Jour. Med. Sciences*, April, 1895), and search for some peculiar bacterium, or toxic, or auto-toxic element, but what divides acute mania from chronic mania, or either from the exacerbations of inherited defective states. Indeed, what separates mania from melancholia? Is it the simple emotional bent, trivial and changeable? What separates paranoia from chronic mania, or melancholia? Surely, it eludes our finding at times. What separates senile dementia from other forms or from previous sanity? What separates any form from "sanity"? Is not the time fast going by when we place all men in two classes--the sane and perfectly responsible, or the insane and perfectly irresponsible? Have not the sane varied degrees of mental ability and stability; have not the insane as well? Are not the above names simply to designate "types," quite arbitrary and convenient, yet with no power like that of the disease *measles* to compel conformance to special lines.

But there are other things to be wondered at. Among the many ill-defined and strange phenomena is one which seemingly has not attracted the attention that it deserves. This is "periodicity." As *folie-circulaire* it quite early received some study, and has a place in some of the asylum reports. At first assumed as a peculiar and rare entity, it now seems only an extreme form of an element very common among the insanities, and which throws an uncanny air of mystery and inevitableness over their ways. A man whom we have seen almost daily for the past ten years, has, for over twenty-five years, gone slowly into a dull, quiet and rather stupid melancholia, to stay for some weeks or months, only to come out some day, within twenty-four hours, as an excitable, slightly exalted, boisterous, garrulous and active talker. From four to eight times a year this has been kept up. No progressiveness in character, nor tendency toward dementia is noted. His acuteness is in some ways great, his memory quite remarkable. His mental power is at times near normal. A woman is now under observation who for thirty-seven years has had like vacillations.

A year ago, a young man oscillated between a slight

elation with somewhat tipsy-like, silly behavior and a fairly sane state. After four or five oscillations he was able to keep his sane standing and was sent home. He remembered everything and was closely questioned. But he said simply that he felt like a tipsy or drunken man while in the spell, and could give no starting cause. He wanted us to give one. Could we solve the riddle for him?

One of the most common of these periodicities occurs with the menstrual function, forming at times a quite true "circular insanity." Even this does little more than afford a hint, however, of an unstable mind, overturned by the nervous and morbid excitation, such as described by Krafft-Ebing as accompanying menstruation. Many of the cases among women have no cause known. It is true, of course, that the periodicities of malaria, of migraine, of epilepsy, furnish some previous experience to blunt our sense of wonder at these things. Such wonder is still farther blunted by noting, after considerable experience, that such cycles may blend with the ebb and flow of sleep and waking, or of winter and summer, or the less regular ones of fatigue and rest; or (in inebriates) of sprees and sobriety, and as well of invasion and recession in fevers and other bodily diseases. And yet the wonder is great. The circulation seems at times affected, but seems wholly insufficient to account for the phenomena.

Then again the recovery of chronic cases is at times startling. To see a person after two, three, five, or even ten years of mental darkness, quite suddenly become clear and intelligent is a mystery like that of birth and hardly analogous to sleep. If so easily slipping back to normal, where was the lesion, and why could not recovery have happened sooner? What was the cause of its happening now? Can cells and fibres for long years have their action annulled only to spring at once into their old activity and the cause be unknown? After all the changing vicissitudes, surroundings, medicines, and other causes of years, with seemingly no new element, the case may quite abruptly change toward recovery. The studies of Hodge, of Andriezen, of Ramon y Cajal, of Berkeley and others, hardly helps us here, though they keep up our hope of ultimate solution.

**Dr. Daniel Clark**, of Toronto, is the author of a modest little manual on the subject of "Mental Diseases." It is of more peculiar value as coming directly



from a practical experience among the insane. It is plain, practical, and unencumbered with the technical and unusual terms often so needless in actual work. It is evidently for medical students and as such will be of high value.

In this manual he excludes idiocy, imbecility, hysteria, hypochondriasis, decay of old age, toxic effects of opium and other drugs, crankism, eccentricity, and the deliriums of fever from his definition. The definition is as follows: "Insanity is a fixed physical disease, which affects and controls abnormally the language, conduct, and natural characteristics of the individual."

No very precise classification seems attempted. Puerperal insanity has a chapter by itself, instead of a discussion of "puerperal" among the causes. Circular insanity is discussed by itself in an interesting way. "Moral insanity" is affirmed, and a state described in which moral change is symptomatic of brain disease due to such causes as sunstroke, traumatism, etc. He guards against including much of criminality, and also separates the class from "moral imbecility" which latter probably is in ordinary discussion most often designated by the former term. "Paranoia" he holds to be "not a distinct phase of insanity," even while giving "puerperal insanity" and "phthisical insanity" no such qualifying words.

Some miscellaneous chapters follows. One on "responsibility" is very interesting, as also some words on medico-legal testimony. Haematoma auris he rather upholds as non-traumatic as against the views of Matthew Field. Alienists will find this book very interesting reading.

**Dr. Richard Dewey**, formerly superintendent at Kankakee, Ills., and recently in practice at Chicago, where he has also been acting as editor of the *Journal of Insanity*, has taken charge of the Sanatorium at Wauwatosa, the superintendency having been made vacant by the resignation of Dr. McBride. By this is also probably indicated the permanent disappearance of the *Review of Insanity and Nervous Diseases*, which has been so successfully edited by Dr. McBride. All good wishes are due Dr. Dewey in his new work.

**New appointments.** The following newspaper clipping reports briefly the changes made by the new Board of Control in Wisconsin, a change of political parties having occurred at the last election:

MADISON, Wis., June 12.—The state board of control has made the following appointments: Warden of state prison, J. J. Roberts, Waupun; state hospital for the insane superintendent, Dr. William D. Lyman, Galesville; matron, Elizabeth Whitehead, the present incumbent; steward, Charles A. Carter, Milwaukee; Northern Hospital for the Insane: Superintendent, Dr. A. W. Gordon, Oshkosh; steward, E. Finney, Oshkosh. School for Deaf at Delavan, John W. Swiler, present incumbent, reappointed superintendent. State public school: Superintendent, S. S. Landt, Friendship; state agent, Peter Williams, Portage; matron, Mrs. Jennie Brewer, Baraboo; teachers, Maud Utter, Nellie Jones, Anna G. Monahan, Nellie Hankin, Florence Parry, Edna Jones, and Clara McMillan.

*The Medico-Legal Society of New York* is aggressively invading the field of alienistic work, as the proceedings of its congress held in New York City, September 4 to 6 inclusive shows. It has been very widely noticed by the public press. Workers among the insane will look with interest for the printed form of the articles which were there given.

## Periscope.

### PHYSIOLOGICAL.

**On Little Known Special Manifestations in Nervous Diseases, and on the Diagnostic Significance of the Ankle Clonus and of the Variations in Tendon and Cutaneous Reflexes.**—Bechterew (*Neurologisches Wiestnik*, 1895, Vol. iii., No. 2).

1. The first phenomenon described concerns hemiplegics.

If the hemiplegia is such that the patient is not able to perform any voluntary movements with the paralyzed arm, the following phenomenon can be frequently produced. When the forearm of the paralyzed side is passively flexed ad maximum on the arm and is then extended by rapid force, the extension does not take place uniformly as under normal conditions, but, arrived at a certain position, the forearm suddenly stops in its downward movement or even rebounds; the continuation of the extension until the forearm is in a straight line with the forearm occurs normally without a repetition of the phenomenon described. The latter is causally connected with the rapid distention of the biceps muscle.

2. In some cases of multiple neuritis and of tetany B. has noticed a retardation of the knee jerk, that is, the latter would not take place immediately after the tendon was hit, but some time would elapse between the stimulus and the response; yet in force the knee-jerk was not diminished. If in such a case the tendon was repeatedly percussed with the hammer for some time, the knee jerk diminished in force and finally disappeared. When after some rest the knee jerk was tried again, it was found to have reappeared, but was still more retarded than at the beginning of the test. The author compares the phenomenon with the retardation of the cutaneous reflexes met with in locomotor ataxy.

3. In cases of motor and sensory paraplegia, chiefly in those of spinal, occasionally also in such of cerebral origin, the following reflex was sometimes observed: Slight and uniform percussion of the anterior side of the tibia was followed by periodic contractions in the muscles of the thigh, occurring repeatedly at regular intervals (no less than every four minutes, at the most every ten to twelve minutes, on the average every six to seven minutes). Sometimes these contractions were so intense as to lead to a flexion of the knee. Nothing definite can be said of the diagnostic value of this symptom, apparently it is in those cases which present exaggeration of the tendon reflexes.

4. Discusses the diagnostic value of the ankle clonus and of the exaggeration of the knee-jerk.

a. Ankle clonus:

From observations made by him and other authors B. concludes that the ankle clonus is not as characteristic of primary or secondary affections of the pyramidal tracts as many neurologists seem to assume. In confirmation of Gowers' observations, he has seen the occurrence of ankle clonus in hysteria. He also confirms Westphal's statement that it is sometimes present in cases of chronic articular rheumatism. Aside from this B. has seen this phenomenon in cases of myoclonus multiple, of akinesia algida, occasionally also of multiple neuritis. The fact that in these cases the clonus was most developed when the disease was at

its height and diminished when the disease subsided proves the causal connection with the latter.

*b. Unequality of the knee-jerks.*

The question is discussed whether unequality of the knee-jerks, that is, a difference in force and promptness between the knee-jerk of one and that of the other side, can be considered as a sign of organic nervous disease. The question is answered in the negative, as this unequality is found also in functional nervous diseases, viz., in akinesia algèra, myoclonus multiplex, hysteria and neuralgia (for instance, neuralgia of the sciatic nerve; it is true that here the neuralgia may sometimes be organic). Inequality of the knee-jerk may also be met with in chronic articular rheumatism when the disease is localized in the joints of one side only. In hysterical palsies the tendon reflexes are frequently increased on the side of the paralysis. When hemianæsthesia is associated with it, the cutaneous reflexes are diminished in contrast to the increase of the tendon reflexes.

*5. Treats on cutaneous and other reflexes as influenced by sensory disturbances.*

Anæsthesia causes lessening, hyperæsthesia exaggeration of the cutaneous reflexes. Aside from the exaggeration of the cutaneous reflexes, there are other signs that can help us prove the existence of hyperæsthesia in doubtful cases, viz :

*a.* The production of the pain reflex of the pupil by means of sensory irritation of so slight degree that under normal circumstances it would not cause any pain.

*b.* The vasomotor effects (rushing of blood to the head, acceleration of the pulse, change of the pulse curve), the vertigo and the alterations of the character of respiration called forth by pain stimulus of quite mild degree (pinching, stinging).

These symptoms often seen in hysteria and traumatic neurosis are of diagnostic value as they make it easier to make the differential diagnosis between said neuroses and simulation.

ONUF.

PATHOLOGICAL.]

***The Posterior Corpora Bigemina as Centres of Hearing, of the Voice and of Special Movements.***—Bechterew (*Neurologischer Wiestnik*, 1895, Vol. iii., No. 2. Russian).

After discussion of the anatomical investigations which have proven the connection of the posterior corpora bigemina with the eighth nerve, the author passes over to a report of the results produced by ablation (and excitation) of these masses of gray substance. The experiments were performed upon rabbits, guinea pigs and white rats. The following conclusions are reached :

1. If the destruction of both posterior corpora bigemina was sufficiently thorough, a more or less marked diminution of hearing or complete deafness followed.

Superficial destruction of the gray masses mentioned did not have any noticeable influence upon the sense of hearing of the animals.

2. Where the ablation had been sufficient, it was noticed that besides the impairment of the sense of hearing, a weakening or even complete cessation of the voice was the consequence. Unilateral ablation caused only weakening of the voice. Superficial ablation had no effect upon it.

3. After thorough ablation of the posterior corpora bigemina the animals lose the faculty of standing and gait, although otherwise the motility of the extremities is preserved.

The symptoms observed during the first hours after the lesion of the posterior corpora bigemina were: Staggering gait, forced movements or forced positions, sometimes manège movements with devia-

tion of the eyes, lying upon one side of the body, turning of the head, occasionally nystagmus, etc.

Similar motor disturbances, only in a reversed sense had been observed after excitation of the posterior corpora bigemina.

Although the existence of a cortical centre for the voice has been claimed and described by Krause, Simon, Horsley and Masini, the author found that there is no spot in the cortex whose destruction would have any influence on the voice. Transverse (frontal) sections of the brain anywhere cephalad from the corpora bigemina produce no alteration of the voice; when the section is made caudad from the corpora bigemina posterior, the voice ceases altogether.

The author does not believe that the effect upon the voice and the other motor disturbances observed as a sequel to ablation of the posterior corpora bigemina is due to lesion of deeper lying structures, for instance, of the superior cerebellar peduncles, but is convinced that the posterior corpora bigemina are themselves the centres for said movements.

ONE.

#### CLINICAL.

**On a Symptom Complex of Congenital, Apparently Acquired, Disturbance of Co-ordination.**—Nonne (*Archiv f. Psychiatric u. Nervenkrank.*, Vol. xxvii., Part 2).

After referring to his first publication on this subject in which he portrayed the symptom complex of a peculiar family disease as it had manifested itself in three brothers, and in which he gave the findings of a careful histological examination of the nervous system in one case which had come to autopsy, the author takes a brief retrospect of the contributions that have been made to this subject and conditions simulating it and then proceeds to put on record the histories of six cases.

A synopsis of these cases shows:

In case No. I.

1. Spontaneous (or possibly after measles), early beginning (five years old).

2. No heredity.

3. Disturbance of co-ordination, involving the muscles of the extremities, of the trunk and the mimic muscles.

4. Severe involvement of the lower extremities.

5. Abnormal liveliness of the tendon reflexes.

6. Tendency to rigidity of the muscles of the lower extremities.

7. A "phonetic" disturbance of the muscles of the lips, tongue and larynx which subserve the act of speech.

8. Slight insufficiency of the external eye muscles and manifestations of nystagmus.

9. Slight deterioration of intelligence. No disturbance of the pupils; no alteration of sensibility; sphincters intact; no atrophy of optic nerve.

In Case No. II.

1. Spontaneous and early beginning (seventeen years old).

2. No heredity.

3. Comparatively severe disturbance of co-ordination in the upper and lower extremities simulating "cerebellar" ataxia.

4. Liveliness of tendon reflexes without muscle contracture.

5. Phonetic disturbance of speech and concomitant affection of the mimic musculature.

6. Limitation of intellectual capacity.

7. Slight insufficiency of external eye muscles; no nystagmus; no optic atrophy; no disturbance of pupils.

8. Normal sensibility and sphincters.

9. Chlorosis and muscular symptoms.

In Case No. III.

1. Early and spontaneous beginning (five years old).
  2. No similar case in the family. Two other children seem to have had encephalitis.
  3. Static ataxia and cerebellar gait, and slight disturbance of co-ordination in upper extremities.
  4. Increased tendon reflexes and tendency of the muscles to rigidity.
  5. "Explosive" manner of speaking.
  6. Slight insufficiency of single external eye muscles. No nystagmus; no optic atrophy; no disturbance of pupils.
  8. Intelligence normal.
- In Case No. IV. the symptoms developed in a child seven years old, after it had an attack of measles.

The 5th and 6th cases which the author publishes are meant to demonstrate that a similar symptom complex may be the residuum of an acute disease of the brain.

Case V.—Male, 20 years old, without family or hereditary predisposition, developed in his twelfth year after the termination of a disease in which cerebral symptoms predominated:

1. A locomotor disturbance of co-ordination of the four extremities, which varied from an ataxia to a tremor. Static ataxia.
2. Speech disturbance of a phonetic character, dependant upon a similar inco-ordination of the speech respiratory muscles.
3. Insufficiency of the external eye muscles, normal pupils and normal optic nerve.
4. Moderate defect of intelligence.
5. Liveliness of tendon reflexes; slight muscular rigidity.
6. Absence of sensory or bladder disturbance.

Case VI. is very analogous to Case V.

The author's conclusions are as follows: The disease developed in these patients without any hereditary condition existing in the family. It may follow a disease of the brain, as we have seen it after a sun-stroke. The most important symptoms are:

1. Ataxia of station and locomotion, ataxic inco-ordination of the extremities (not pure), of the trunk and phonating speech muscles, with intact power of mimicry.
2. Insufficiency of the external eye muscles, with normal condition of the pupils and absence of nystagmus and change in the optic nerve.
3. Increase of patellar tendon reflex without accompanying rigidity of the muscles.
4. Absence of disturbance of sensibility and absence of sphincter disturbance.
5. Intactness of the intelligence.

The symptoms in one of the patients which the author details reached a standstill. Nonne is of the opinion that we have in these cases to deal with the sequences of an affection of the brain, the result of some toxic agency, of exogenetic origin.

He thinks it possible also that further experience will show, now that attention has been called to it, that the symptom complex here described is not so infrequent as one might think, and especially that isolated cases will be found in families in which there is no other nervous trouble.

J. C.

**Chloroform Anaesthesia Produced During Sleep.**—Dr. R. Gurriere (*Rivista sperimentale di freniatria, etc.*).

G. has repeated the experiments of Dolbean, the purpose of which was to find out whether chloroform vapors could be administered to a person physiologically asleep, and whether anaesthesia could thus be produced without awakening the sleeper. While Dolbean had experimented upon individuals affected with somatic diseases, G. performed his experiments upon persons who, being perfectly healthy somatically, had

passed through some mental disease but were cured so that their discharge could take place a short time after the date of the experiments.

The technic was to begin the administration of the chloroform very cautiously. A handkerchief drenched with chloroform was first kept about three inches from the nostrils then gradually approached. If reflex movements or wiping off the nose with the hand followed a short interruption was made. The patients were not informed what was going to be done with them, all possible precautions were taken not to make them suspect anything.

The result of the experiments was positive in four cases out of nine examined ones. In one of the five cases with negative result it had been observed that the cause of the failure was probably too light, superficial sleep. The precaution was then taken to give this person, without his knowledge, one-twelfth grain of morphine some hours before bed time. The chloroform narcosis was then tried again during sleep and this time successfully, so that actually the result was positive in five out of nine cases.

The narcosis was pushed only so far that it could be ascertained that the subject in question was anesthetized. Although it could have been done, the author did not want to reach the degree of anesthesia required for surgical operations, he pushed the narcosis so far, however, that no doubt could be entertained as to its anesthetizing effect, the limbs were perfectly relaxed, flapping the body and making noise did not awake them, only by strong shaking they finally awoke, but did not remember that they awoke during the night when asked the next morning, and had not the least idea that chloroform had been administered to them.

The result of the experiments is important from a medico-legal point of view, as it proves the possibility of anesthetizing a person during sleep for criminal purposes. ONUF.

**Trinitrin in the Treatment of Sciatica.**—(*Journal de Med. de Paris*, April 21, 1895).

Dr. Mikhalkine, of Nijni-Novogorod, has had occasion to test the anti-neuralgic properties of trinitrin (nitro-glycerine) in three cases of inveterate sciatica which showed themselves absolutely rebellious to such remedies as antipyrine, phenacetine, acetanilide, chloral hydrate, the bromides and other analogous preparations.

Under the influence of nitro-glycerine two cases were radically cured of their sciatica, and in the third case marked improvement took place. The trinitrin was administered either in the form of a one per cent. solution in alcohol, of which the patients took three drops daily, or in the following mixture :

R	1% alcoholic sol. trinitrin,	5.0.
	Tr. Capsici	7.5.
	Aq. menth.	15.0.
Dose.	5 to 10 drops, three times daily.	

MEIROWITZ.

**Acromegaly.**—E. L. Bullard, M.D. (*The Med. and Surg. Reporter*, April 27, 1895).

After discussing the history, symptomatology, pathology, and differential diagnosis of acromegaly, the author gives the history of a woman affected with this disease, who presents the following characteristics: The ears, nose, lower lip, tongue, inferior maxilla, hands, feet, clavicles, ribs, sternum, patellæ, and pelvic bones are enlarged. There is prognathism to the extent of about one-half inch. The neck is short and thickened; the thyroid gland is enlarged and cartilaginous, the right side thereof being the larger of the two. There is pulsation in the veins of the neck; cervico-dorsal kyphosis is marked and increasing. Her former weight was 127 pounds, it is now 214. Her height is five feet, and the length of the foot (which ordinarily bears the relation to the height of one to six) is eleven inches, or one to five and a half.

There is impairment of sight, but the eyeballs are not as prominent as they were several years ago. The eyes are surrounded by a pigmented areola, which varies in intensity. The mental faculties are little, if any impaired, but the memory is poor. Polyphagia and polydipsia are present. There is hyperidrosis, saccharine urine and albuminuria have at times been observed, and there has been considerable œdema of the feet at times. The voice is masculine. MEIROWITZ.

#### THERAPEUTICAL.

**Tea and its Effects.** Jas. Wood, M.D. (*Quarterly Jour. of Inebriety*, April, 1895.—"Theinism," according to this author, is a common condition, for he found that since January 1, 1894, out of 1000 patients applying for treatment, one hundred gave symptoms of tea inebriation. The theine affects the nervous system primarily and the organic system secondarily; the taumic acid affects the digestive apparatus and organs connected with it, while the essential oil gives the peculiar intoxication so typical of tea dipsomania. The length of time tea is infused will greatly change its composition and also its action upon the system. In an infusion of fifteen minutes of the finest Indian tea, the yield of taumin is nearly two and a half times as much as the finest china. In all teas the length of time of the infusion affects greatly the composition, with possibly an exception in the case of the better qualities of China tea. The essential oil is present in larger quantities in the first infusion than in subsequent ones and if the tea is not drunk immediately it is soon lost. This explains the headaches of professional tea-tasters, who use the infusion immediately after it is made. The amount of tea which can be taken with impunity differs with the individual, but usually it is found that an ounce of tea-leaves used daily will soon produce poisonous symptoms. This amount would contain about ten grains of theine. From the author's 100 cases the following analysis of symptoms was prepared:—Sex, sixty-nine per cent. female and thirty-one per cent. male. Quantity, two pints or less, fifty-four per cent; four pints or less, thirty-seven per cent.; ten pints or less, nine per cent. Strength, seventy-seven per cent strong, fifteen per cent. ordinary, eight per cent. not known. Number nervous, seventy-two per cent. Bowels, forty per cent. constipation, two per cent. diarrhœa, fifteen per cent. irregular. Pains, sixteen per cent. genual, ten per cent. heart, nine per cent. back, six per cent. side, seven per cent. chest. Dizziness, twenty per cent. Faintness, eight per cent. Gastro-intestinal indigestion, nineteen per cent. Dreams, five per cent. Depression, twenty per cent. Excited, five per cent. Suicide, three per cent. Headache, forty five per cent. Palpitation, nineteen per cent. Muscular tremor, twelve per cent. Insomnia, fifteen per cent. Theine which is the most important constituent of tea, lessens tissue metamorphosis, and if pushed far enough causes general arterial excitement, tremor, and a very urgent desire to urinate. The imaginative faculties become more acute or the mind may wander. It seems to affect chiefly the sensory system, but in larger doses may come convulsions. The amount of nitrogenuous elements found in tea is too small to have any nutritive value. The essential oil is found more plentifully in the green tea and seems to be lost during the oxidizing process through which the leaves are put to produce the black variety. Green tea is also richer in theine and taumin. The influence which the green tea has on the nervous system and for which it is largely noted is due to the essential oil. The better class of people drink black tea, while the poorer classes, in Ireland especially, use the cheaper varieties and cannot avoid the deleterious effects which the better class escape. In reports just received from Ireland great prominence is given to the immoderate use of tea as a cause of insanity. The difference in the sexes, regarding the consumption of



tea, is probably explained by the greater use of tobacco by men and the consequent satisfaction for a stimulant, while women assuage the importunities of the system for a stimulant by tea. While theine and caffeine produce diametrically opposite effects for the most part, they are similar in causing cerebral excitement, wakefulness, hallucinations and a soporific state following the exhaustion of insomnia. Tea and coffee undoubtedly antagonize each other, or rather the symptoms which each are likely to produce alone are not present when the two are used together. This was noticed in some patients who could drink large quantities of tea and coffee and be but slightly affected. They, however, complained the most of insomnia and excitement and of almost no other symptom. FREEMAN.

**Therapeutic Effect of an Exploratory Craniectomy.**—Dasara (*Clinica Chirurgica*, 1895, p. 35).

As the result of a fall from a horse, the patient presented all the signs of fracture of the skull; coma, otorrhagia, delirium, etc.

This condition lasted a week, when epileptiform convulsions made their appearance and became more and more frequent. There was nystagmus, deviation of the eyes to the left, contracture of the left side of the face, trembling of the left arm and leg, trismus, and at last tetanic spasms of the whole body. Respiration stertorous; lack of reaction in the pupils; face cyanosed; pulse rapid; axillary temperature 100.

The attacks increased in frequency until finally they came every two minutes. In a few hours the temperature rose to 103.

A large exploratory incision at the point of the right asterion was made, but nothing abnormal was found either upon the meninges or in the brain. The attacks having disappeared as soon as the skull was opened, nothing further was done. The results of this operation were remarkable; the temperature immediately returned to normal and the attacks ceased. At the end of twelve days the patient was entirely himself again. In view of this unexpected result, Roth was of the opinion that the lesion was cerebral contusion, fracture of the internal table, and traumatic meningo-encephalitis, which was arrested in its evolution by the craniectomy. ROBINSON.

**A Case of Tetanus Cured by the Antitoxin of Tizzoni-Cattani.**—Caretti (*Riforma Medica*, 1895).

Seven days after the reception of a contused wound of the head, trismus appeared without fever.

Chloral and bromide of potash were given, but the disease grew steadily worse.

Injections of antitoxin were made forty-eight hours after the attack. These were repeated morning and evening for four days, and the patient continued to take the chloral and bromide.

The condition remained the same for two weeks, then the facial paralysis and the trismus progressively disappeared. The temperature never went beyond 103. This is the second case of tetanus treated by Tizzoni's antitoxin. ROBINSON.

**Insomnia in Surgery.** G. G. Van Schaick, M.D. (*A. E. Med. Jour.*). Insomnia from whatever cause is an important complication of surgical disorders. Its relief is necessary to the patients' comfort, and thereby the prognosis is improved and recovery materially assisted. Where pain is the chief factor, morphine is the only drug that will relieve with certainty. There are many surgical disorders with insomnia which may be relieved by trional. This remedy acts rapidly and safely, has no inhibitory action upon the secretions, seems to possess a stimulating effect, is well borne by the stomach, easily absorbed by the rectum and produces no unpleasant after effects. FREEMAN.

## Correspondence.

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*To the Editor of the JOURNAL OF MENTAL AND NERVOUS DISEASE.*

Sir:—In the August issue of your valuable Journal your correspondent, Dr. E. P. Hurd, refers in a critical vein to some portions of a paper of mine published in the July number of the same Journal. The paper is an abstract of one on "Duplex Personality—Its Relation to Hypnotism and to Lucidity." I will endeavor to reply to his criticisms in the order in which they are presented. He says: "I read the following astonishing statement anent subliminal possession of supra-normal perceptive powers." "Alleged facts of this kind are not received by the strictly physiological school of psychologists." "I write to inquire whether this is a 'fact?' Have we a school of psychologists who are not willing to receive facts?"

I reply first: There certainly is a school of psychologists who do not receive *alleged facts* of the kind which I adduce. The late Dr. Carpenter (*Mental Physiology*) and Wundt (*Psychology*) are examples of this school. As examples of the alleged facts which they do not receive I will simply adduce clairvoyance. No one, I think, will accuse either Carpenter or Wundt of receiving this alleged fact, and Dr. Hurd himself with all his interest in the subject does not hesitate to say that he would require facts of an uncommonly well attested kind before he "could believe in the possibility of visual perception gained where vision by the physical organ of sight is impossible." Evidently he does not accept the *alleged* fact. I can hardly see, therefore, why my statement that "alleged facts of this kind are not received by the strictly physiological school of psychologists should be considered an 'astonishing statement.'" What Dr. Hurd really means is that the psychologists are all right, but that the alleged facts are not—and it is my statement that these alleged facts "are just as well established as any other fact in nature," that he really intends to characterize as "astonishing": and on this point the fine scorn and *nez retroussée* are not surprising. They are simply amusing.

If my memory serves me several alleged facts in nature received similar treatment when they were first presented for consideration. There was a certain Galileo, a Newton, a Franklin, a Darwin, and several other well-known persons who suffered some personal inconvenience and abuse at the hands of the representatives of accredited science because they brought forward "alleged facts" which were not consistent with the theories of nature then held as orthodox. The people who thought it their duty to stand up for orthodox science, as they understood it, were not altogether to be blamed—they were made so and educated so;—they knew several things, but they could not see that the earth moved, that gravitation controlled the movements of the planets, that lightning and electricity were one and the same thing, or that new species were developed by minute changes fostered by new and favoring environments; there was no place in the texture of their thought for these new facts, and it took a generation or two in each case for men to get the texture of their thought changed and improved so that such ideas could be entertained. Then the fine scorn subsided, the *nez retroussée* relaxed and the "alleged facts" quietly took their places as accredited

science. The same thing is being repeated to-day regarding unusual psychic phenomena of which clairvoyance is an example.

This is hardly the place to enter into an elaborate proof of the fact of clairvoyance, but I may be permitted to present a brief outline of the argument.

The proof is three-fold :

First, there is the historical argument. Every nation or people which has a history or a literature has accounts of persons gifted with clairvoyant perception. Hebrew literature shows numerous examples ; it was even a part of the office of their priests and prophets. Pythagoras a century before the time of Socrates found this faculty made use of in Egypt, Assyria, Arabia and India. Greek and Latin literature whether in prose or verse, history or the drama abounds in examples. Homer, Xenophon, Virgil, Tacitus, and numerous later writers furnish hundreds of instances clearly described, while in our own era literature abounds in such examples.

Second, there is what may be called the anthropological argument. It shows that nearly every people, whether civilized or barbarous over the whole surface of the globe, however widely separated and in however widely differing circumstances of climate, comfort or culture, point with assurance to cases of clairvoyant vision amongst themselves, and did space permit most interesting examples could be adduced from each different locality. Not only does clairvoyance occur in all these widely separated countries and localities, but it occurs under similar mental conditions, and when artificially produced similar means are employed. Is it probable that the human mind is so constituted that it should work out identical frauds, and by the same methods and concerning this one strange and unusual subject in places so widely separated from one another and under such strangely varying conditions ?

As proof of so strange and unusual a phenomenon as clairvoyance, however, these arguments alone might be rejected as insufficient, if they were not corroborated by direct testimony and present observation ; but they surely prepare the way and add force to recent and direct proof—and such proof is not wanting. Not a few, but hundreds of cases have been thoroughly observed and carefully studied by competent, shrewd and trustworthy men. Amongst those who have reported cases I may mention Profs. Charles Richet and Pierre Janet, Drs. Azam, Dufay and Gilbert, of France, and Dr. Backman, of Sweden ; excellent cases have also been reported to the English Society for Psychical Research, and I may add that after many years of waiting I have myself had an excellent example under my own observation within the past year. These cases would fill a volume. In studying them all paid or professional clairvoyants are left entirely out of the count. In some cases the clairvoyant faculty may have been exercised only once in a life time, in others almost daily for years ; some while in the condition of natural somnambulism, others only when in the hypnotic state ; with others it has occurred in connection with crystal gazing and in the condition of abstraction or reverie.

Neither is this the place to discuss the fact nor the characteristics of the subliminal self ; nearly all intelligent observers, however, in this field of psychology know that it is *something* with all the characteristics of a well-marked personality, differing in many important particulars from the ordinary personality ; they also know that the " alleged fact " of clairvoyance appears in connection with this subliminal personality, hence I consider it fair to infer that the faculty of clairvoyance, or of a superior or supra-normal perceptive faculty, by whatever name one may choose to call it, is an attribute of the subliminal self. As stated in the abstract which has been made the subject of criticism, I do not assume to dogmatize upon this point. Nevertheless I think those psychologists are wise who learn to look upon the subjects of clairvoyance and the

subliminal self from this standpoint, as well as from that of the strictly psychological school of psychologists with their "unconscious cerebration" and "expectant attention," or their purely cerebral explanation based upon "clogged cells," "well worn nerve paths," "inhibited tracts," etc.,—conditions which are altogether inferential and have never been observed either with or without mental phenomena.

Such is an outline of proof. Many intelligent men have followed it, have added personal observation to the other proofs, and are just as well convinced of the reality of clairvoyance as of "any other fact in nature." Others doubtless have examined the proofs according to their best light and still are unconvinced. I am not blaming such persons—they are acting in accordance with their idea of what true science is; doubtless they have done good work in some important department, but may it not be possible that there are other departments of science in which they are not equally intelligent and in which they are not equally well constituted for efficient work. My "allegations" were certainly not intended to "hit" these persons in any uncivil way, nevertheless I believe my "alleged facts" will stand.

R. OSGOOD MASON,  
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THE  
Journal  
OF  
Nervous and Mental Disease.

Original Articles.

ON THE MORBID HEREDITY AND PREDISPO-  
SITION TO INSANITY OF THE  
MAN OF GENIUS.<sup>1</sup>

By WARREN L. BABCOCK, M.D.,

Assistant Physician, St. Lawrence State Hospital, Ogdensburg, N. Y.

“**M**EN of genius are instruments on which the melody of nature, like sphere music, was made for the benefit and delectation of such as have ears to hear.”<sup>2</sup> A more graceful description of the genius would be hard to find in the annals of literature. Maudsley, whose delightful prose has been the envy of many a versatile writer, here aptly expresses in a few words his appreciation of the most gifted class of men, and, when compared with a labored psychological interpretation its beauties will be still more appreciated. In the language of the student of the mind, the quality of genius may be defined as “An exalted mental power, combined with a special instinctive aptitude independent of tuition.” The term genius has long enjoyed a wide latitude of application, and it becomes necessary before the men of great gifts can be properly studied or their characteristics rightly defined, to divide them into two primary classes. *First*, the normal or regenerate, and, *second*, the abnormal or degenerate men of genius.

The normal man of genius can be dismissed in few words. He is now the highest type of our present state of

<sup>1</sup> Read before the Binghamton Academy of Science, Feb., 2, 1895.

<sup>2</sup> Maudsley, *Physiology of the Mind*, p. 64.

evolution and civilization; he is the talented man of our colleges and universities, the leader of his profession and the director of all movements of progress. By force of industry and tact he has acquired those resources of mind which characterize, in larger measure, the greater genius. A superficial comparison would reflect him as a copy in miniature of his degenerate cousin, but a deeper study would establish a wide divergence, both mental and physical. Well developed qualities of originality, attention and abstraction are strangers to his mentality; his heritage bespeaks a normal ancestry and his symmetry of physical contour a healthy organism. The work of the mediocre or even the talented man fades away in the march of ages and sinks into obscurity. A generation or two suffices to obliterate his labors from the history of mankind, and though not having lived in vain, he lived for his time and generation only. But the discoveries of really great men never leave us; their works live on and on and their fame proves immortal.

The study of the abnormal man of great natural gifts is a problem which is as varied as it is interesting and its results as conclusive as the most skeptical could wish. Undoubtedly, the preponderance of great natural gifts of mind, which in themselves insure great renown, entails a corresponding deficiency in other parts of the mental and physical economy, and though in many respects the man of genius may well be the subject of envy, he may in other regards, as consistently be the object of pity.

The study of this subject involves the comparative consideration of the other varieties of the degenerate elements of the race. The protean divisions of degeneracy comprehend a diverse array of nature's anomalies. The one essential characteristic of this morbid group is the hereditary factor which alike influences each division of the subject. The taint is beyond the power of the individual to rectify, and a faulty ancestry is responsible, atavistically or otherwise, for the perverse character of the members of this group. Their hereditary, adolescent and adult history, together with their remarkable mental and physical resemblance, relegate the genius, the instinctive criminal, the idiot and imbecile, the deaf mute, and some classes of the insane, notably the paranoiac, into a group whose family likeness is striking to contemplate.

Morel's classic division of degeneracy in 1857, was the first attempt to study the subject from a scientific and psychological standpoint. He divided it into five classes from the standpoint of etiology, as follows: toxic, miasmatic, urban, morbid and mixed. His morbid class comprehends all of the primary degenerations mentioned above, and the tendency of modern writers is to restrict the subject entirely to the group here given.

Within a few years after the publication of Morel's work, Francis Galton drew attention to the subject in its relation to natural selection and heredity.<sup>1</sup> The impetus thus given to the study of degeneracy soon created widespread interest among psychologists and alienists, and the establishment of the science of criminal anthropology on a firm basis during the following decade, opened the portal and paved the way to a more scientific elaboration of the subject, until to-day its various departments command the time and attention of sociologists the world over.

The heritage or family history of the gifted man is a most interesting and complex study. In many cases he is a descendant from an illustrious stock or is a member of an eminent family, as shown by Galton. The Darwin and Herschel families in England, and the Adams, Beecher and Field families in this country, well illustrate this mode of descent, as they have all sent forth generation after generation of illustrious sons, whose works and fame have been world-wide, and who need no obituary in brass or marble to perpetuate their name. But families of geniuses are comparatively few, and the number of illustrious examples of this mode of descent necessarily limited. How much oftener is it stated in history or biography that the origin of this or that grand genius is obscure and worldly; that the clouds and mists of his early life were cleared away by his own indomitable energy and power of application, and that he, early surrounded with obstacles innumerable, arose from darkness and obscurity with as full and grand a glory as attends the rising of the sun.

Again, he may be a degenerate growth from a perverted family stock, inheriting his tendencies from a remote ancestry by atavism, or else one of a large immediate family of degenerates.

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<sup>1</sup> Morel. *Treatise on Degeneracy*. Paris, 1857.

<sup>2</sup> *Hereditary Genius*. London, 1869.

You have all noticed the odd boy of the family; the doubtful character; the precocious prodigy; the black sheep of the flock. For example, let us take a respectable farmer's family of four or five children. The parents are healthy, sober, poor but respectable people. The children, with one exception, resemble one or the other parent. Their mental endowments do not exceed mediocrity. But this odd boy—this variation from the family likeness, is the one who interests us. He differs from the others greatly and is willful, perverse, moody, impulsive, and perhaps frail and sickly. Marked precocity is his only redeeming characteristic. The ordinary pleasures of childhood which interest his brothers and sisters have no charm for him. He plays alone or wanders off in the fields, seeking solitude in the passivity of nature. He is such a strange boy. His parents do not understand him, and his associates look upon him with awe. As he reaches the adolescent period, he becomes dissatisfied and restless, reluctantly gaining his parents consent to leave home, or failing in that, runs away and turns his face toward some near-by large city. Here his interesting career commences and if followed up is found to attain one of four terminations. *First*, and most prominent in the order of frequency is an early death.<sup>5</sup> *Second*, he may help swell the criminal ranks. *Third*, he may become mentally deranged and ultimately find his way into a hospital for insane.<sup>6</sup> *Fourth*, and least frequently, he startles the world by an invention or discovery in science or by an original composition of great merit in art, music or literature. He is then styled a genius.

Berti collected records of one degenerate family passing into four generations and found the following list of eighty persons: ten cases of insanity, nineteen of functional neuroses, three criminals, three of great talent, twelve died in childhood, and the family finally became extinct. The original progenitor was an insane melancholiac. The Jukes family in this country forms another good example.

The question now arises—who is responsible for the degenerate heritage? We trace the family history back a few generations. Here in one place is a distinct his-

<sup>5</sup> The tendency of precocious children to die young has become a matter of notoriety. Mercier, *Sanity and Insanity*; p., 179.

<sup>6</sup> Precocious children are liable to nervous disorders, and on attaining adult life are liable to insanity. Mercier, *Loc. cit.*



tory of alcoholism; still more remote, a case or two of mental derangement or perhaps a criminal ancestor. The degenerate or modified taint has been so slight as to only very infrequently crop out, but, nevertheless, we have in the family in a more or less direct line of transmission, a vicious taint. In our hypothetical case it is responsible for the odd boy of the family. In another generation it develops an instinctive criminal, or is the predisposing cause of one or more cases of insanity. "It is undoubtedly true that where hereditary taint exists in a family, one member may sometimes exhibit considerable genius while another is insane or epileptic, but the fact proves no more than that there has been in both, a great natural sensibility of nervous constitution, which from outward circumstances has issued differently in the two cases."

Do not understand me to infer that a genius is born as such. He is primarily a degenerate and as such first sees the light of the world. Physically he is the victim of a number of embryonal vices of development, which in later life become well marked and characteristic and are known as physical stigmata. Mentally he inherits perverted instincts and an exuberant and fertile intellectuality. His abnormally developed, though resourceful brain cells admit of a psychical inco-ordination, such as is manifested in eccentricities, unstable emotions, and delusions of persecution or suspicion. Furthermore, at birth he is a plastic, flexible mold, ready to receive any and all extraneous impressions that nature consigns to his sphere of life. The character of his objective surroundings, and the guardianship of his youth are responsible in a limited measure for the direction in which his talents develop. If the exuberant, fertile soil of his mentality is not cultivated by education and early training, or modified by the civilizing influences of society, it develops along the crooked lines of degeneracy with which it started at birth. Instinctively develops, so to speak, into a foe of social order; while on the other hand, if unfortunate combinations or stress of circumstances prevail, his intellect early succumbs. That the heritage of the genius, either remote or immediate may be better understood, it may perhaps be well to study the subject of heredity especially in its relation to intellectual development.

In the majority of cases, extent or height of develop-

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<sup>1</sup> Maudsley. *Pathology of Mind*.

ment is an index of the intellectual powers. Rapidity of development is accompanied by a corresponding intellectual increase, together with an instability of the higher nerve centres which when of high degree, amounts to mental alienation. This is well illustrated in the mental condition of a person suffering from one of the functional neuroses; by the precocity and the premature arrest of development which occurs in idiocy, imbecility and the degenerate psychoses.

By heredity we mean a reproduction in progeny or off-spring, by transmission or descent, of the salient mental and physical characteristics of the ancestral type. Race after race, individual after individual, have endowed their progeny with a synopsis of the sum total of their objective and subjective experiences plus the similar experiences of their predecessors, whether man or animal. In a more or less varying line they have transmitted down the ages, an impetus,<sup>9</sup> a germ-plasm, a gemmule,<sup>10</sup> a plastidule,<sup>11</sup> a stirp,<sup>12</sup> a micella,<sup>13</sup> physiological units,<sup>14</sup> pangenes,<sup>15</sup> or whatever this potentiality may be termed which has passed from being to being added unto and strengthened in one generation, and weakened or otherwise modified in another, according to the environmental resistance through which it has had to pass.

This hereditary vis-a-tergo, originally primordial in all its salient features, has gradually increased in force and complexity, in harmony with the laws governing natural selection, while this vis-a-fronte, anent the progress made by the application of the laws of science to the resistance of nature, has correspondingly diminished. Thus it is seen that this potentiality which insures, that "like will produce like," when it reaches the individual may be relatively increased or diminished in energy, or otherwise modified by the objective conditions under which it has previously existed.<sup>16</sup> As a rule its most in-

<sup>9</sup> Mercier. *Sanity and Insanity*, 1890.

<sup>10</sup> Weismann. *The Germ-plasm*, 1882.

<sup>11</sup> Darwin. *Origin of species*, Sixth edition.

<sup>12</sup> Elsberg. *Theory of Heredity*, 1874.

<sup>13</sup> Galton. *Natural Inheritance*, 1890.

<sup>14</sup> Nageli. *Theory of Heredity*, 1884.

<sup>15</sup> Spencer. *Synthetic Philosophy*.

<sup>16</sup> De Voies. *Intracellular Pangenesis*.

<sup>16</sup> This theory of heredity is essentially Neo-Lamarckian, and it is based on the transmission of the effects of use and disuse. It originated with Lamarck and has the support of Spencer and Darwin. The opposing theory of Weismannism, more recently developed, has for its sheet-anchor the non-transmission of acquired characters.

delible impress reflects the immediately preceding generation of ancestors, though frequently atavism occurs, and a reversion to a more primitive type is noted.

In the words of Herbert Spencer,<sup>17</sup> "Along with the whole generation of which man forms a part, along with its institutions, languages, manners and multitudinous arts and appliances he is a resultant. . . . All these changes of which he is the proximate initiator have their chief causes in the generation he is descended from."

Each individual act or attribute contributes towards or abstracts from the grand total of this transmitted racial or individual likeness, and in succeeding generations aids in forming the racial characteristics or individual peculiarities of our progeny. From the developing organism this impetus receives a certain acquisition of energy or accumulates new characteristics, which when properly directed and uninfluenced by morbid extraneous agents, insures full and normal development of the embryo to which it is transmitted.

But all theories of heredity must allow for the occurrence of variability, and in this assumption lies the explanation of the degenerate diathesis and atavistic tendency. In terse terms, an individual may start a neurosis or degenerative diathesis in his own life's history, or add to one originated by his parents, or modify an ancestral atavistic vice of development. The prepotent influence of one or the other parent may determine whether the offspring acquire the finely appointed armamentarium of the genius, or the coarse armature of the criminal. When the degenerative element approaches a great degree of morbidity it soon reaches a stage in the family history when the line becomes extinct, thus explaining the well known sterility and barrenness of the degenerate and delinquent classes.<sup>18</sup> Again, the degenerate developmental impetus is misspent or attains so great a degree of velocity as to cause an unnatural diffusion of energy and the individual either fails to properly develop, or else reaches maturity with a damaged and susceptible organism. The accumulation of degenerative vices enables us to explain idiocy and imbecility, while the great rapidity of development readily

<sup>17</sup> Principles of Sociology.

<sup>18</sup> Great law-givers, great philosophers and discoverers in science aid the progress of mankind in a far higher degree by their works, than by leaving a numerous progeny. Darwin. *Descent of Man*. Chap. v.

accounts for the precocity of the genius and many of the insane. His aberrant mental qualities and his physical variations from the normal bespeak a like origin.

Passing from the subject of heredity, we will now consider the relation of the genius to his environment. While all animate beings are more or less in constant warfare with the resisting forces of nature, the relations of the finely organized and abnormally developed individual of great gifts comprehend the two extremes of sublime veneration and marked antagonism. The passivity of nature, resplendent and glorious in its latency, prompts our poets to poesy of the greatest merits. What other influence but close association of mind and heart with the beauties of nature induced Bryant to write,

"To him who in the love of nature holds  
Communion with her visible forms, she speaks a various language,"

or Wordsworth to confess that,

"To me the meanest flower that blows can give  
Thoughts that do often lie too deep for tears."

On the other hand, the activities of nature react powerfully on the genius as though he were the most sensitive flower in the garden of humanity. His sense of environmental resistance is highly developed and acts and reacts to nature's moods with great regularity. Investigation has shown that meteorological, climatic, and racial factors influence degeneracy to a great degree. The maniacal insane are highly sensitive to barometrical variations, being most disturbed during certain states of the weather. The frequency of convulsions in the epileptic is often influenced by the humidity of the atmosphere. The instinctive criminal selects certain seasonable periods in which to ply his vocation. The occurrence of mutinies in prisons bears direct relationship to the influence of the weather on the emotional faculties of the prisoner. When the winter proves unusually severe in India crimes increase from 36 to 48 per cent. We can thus reason by analogy and expect to find the genius influenced by the same natural conditions that disturb his brother degenerates.

A high degree of temperature seems to aid in the elaboration of the best mental work of the man, to whom nature, benevolently or malevolently, bestows great natural gifts. Buffon and Voltaire kept their studies artificially warmed throughout the intense heat of summer. Byron said he feared cold as much as a

gazelle. A well-known English writer wrote while reclining in front of the hearth with his head close to the fire, so close that he often singed his hair. Paisello could only compose between six quilts in summer and nine in winter. Milton's muse was notoriously barren in winter.

From a seasonable standpoint it is noted that men of genius make their most numerous observations, accomplish their greatest discoveries or form their best aesthetic productions in the spring, particularly in the month of May when nature is so resplendent in budding possibilities and so conducive to great endeavors. In his study of genius, Lombroso has shown<sup>19</sup> that during the month of February the productions of the genius are practically *nil* while the spring months, notably May, abound in magnificent works in all departments of knowledge.

As regards climate, it has been found that a mild, warm, sunny clime, like Italy, Southern Germany, or France, produces the greatest number master minds and offers conditions under which the work of the genius is best conserved.

In addition to these factors other influences of a minor import aid in the production and development of the genius. Political struggles, density of population, adaptation of language, elevation above sea level and antiquity of race also manifest influences of great importance in the development of the latent qualities of mind which are the heritage of the gifted.

While a non-resisting environment is often essential to promote the best interests of these sensitive natures, contrawise a condition of effulgence and ease militates against a continued originality and fertility of resource. Necessity has stimulated many an artistic or original production of great merit, while a pension from royalty has reduced the highest talents to mediocrity. Note the marked impoverishment of genius during the reign of Louis XIV.

Buckle,<sup>20</sup> whose philosophical interpretation of scientific and social elements of progress marks an epoch in history writing, says in reference to the royal patronage of Louis, "Men of real genius, great and illustrious thinkers who are the masters and teachers of the human race are thus tricked out with trumpery titles; and after

<sup>19</sup> Lombroso, *Man of Genius*, p. 113.

<sup>20</sup> *History of Civilization in England*, Vol. 1, p. 510.

scrambling in miserable rivalry for the sordid favors of a court are turned into beggars of state who clamor for their share of the spoil."

The subservience of the man of genius to rank initiates a servility of talent to social exactions, and, in the history of many cases a title of baronetcy or a pension from the government has closed the useful career of many a man of great talent.

Let us now study the mental characteristics of the genius. He is the possessor of great fertility of mind; he glories in an instinctive mental aptitude which enables him to employ faculties of observation and attention without appreciable effort. His prolific and bountiful mentality proclaims his exalted mental powers through which his genius finds expression.

He has marvelous powers of ideation and imagination. The concepts of the literary genius may form faster than his pen can transcribe them to paper. "A smaller tendency to imitate goes hand in hand with a greater tendency to evolve new ideas."<sup>21</sup> His imagination is boundless and forms the mental shadow of a reality soon to become manifest in action.

"The genius rejoices in the faculty of imagination which is to be contrasted with the insipidity of the Philistine, and devotes himself with predilection to all sorts of unlicensed pursuits permitted by the unshackled vagabondage of his mind; while he cannot endure well ordered civil occupations requiring attention and constant heed to reality."<sup>22</sup>

Highly developed qualities of attention characterize all gifted minds. In relation to genius, attention may be defined, "As that activity of self which connects all elements presented to it into one whole with reference to their ideal significance."

His emotional faculties are, in common with other degenerates, very unstable and uncertain in their manifestation. A rain storm would drive Schopenhauer to tears and Chopin would weep over one of his own compositions.

He is a man of extreme moods: one moment is exalted, fanciful and filled with brilliant and original conceptions far beyond the scope of the ordinary mind, while the next may find him depressed and melancholy, the victim of an over-developed self-consciousness.

<sup>21</sup> Spencer. *Principles of Sociology*, p. 80.

<sup>22</sup> Nordau. "Degeneration," p. 21.

The pride, vanity and conceit of the gifted but degenerate man is colossal. His boundless arrogance is aptly described by Tocqueville in referring to literary men of talents,<sup>23</sup> "*dans leurs écrits l'auteur paraît souvent grand, mais l'humanité est toujours petite*" Chopin deserted his home and wife, because she offered a chair to a visitor before giving him the invitation to be seated. Balzac when wearing a new dressing gown for the first time wanted to parade the streets with a lighted lamp in his hand.

The genius views the world in free objectivity. His mental endowments permit him to withdraw his thoughts entirely from his objective surroundings and concentrate them upon one subject or line of action. This quality of abstraction, familiar to all great minds, is often complete in degree. A personal knowledge of this condition led Schopenhauer to say that,<sup>24</sup> "The abstraction of genius is a separate, independent existence, a new life which gives its possessor a double personality."

Beethoven would let his hair go several days without dressing and would often promenade the parks and streets deeply involved in abstract thought. Lord Beaconsfield says,<sup>25</sup> "When meditative I am not always sure of my own identity or even existence, for I have found it necessary to shout aloud to be sure that I lived." Hegel calmly finished his "*Phaenomenologie des Geistes*" at Jena on the 4th of October, 1800, without being aware of the battle raging about him. Goethe made observations and experiments for his theory of color during the war in the Champagne amid the bustle of camp life and the thunder of battle. Though his patience was often sorely tried and his life continually in danger, he remained undisturbed throughout. Many more historical illustrations of this mental quality of genius could be given, but the above suffice to well show its character. Indeed, examples of this faculty are so numerous among gifted men that several entertaining chapters might be written on this part of our subject.

The perversity of the genius is expressed by a medley of idiosyncrasies of taste and habit. He may prefer to go hatless or bootless; to sleep on a board or take his meals standing. He turns night into day and day into

<sup>23</sup> *Democracy*. Vol. xiv., p. 139. Paris, 1835.

<sup>24</sup> *Essay on Genius*.

<sup>25</sup> Beaconsfield, Contarini Fleming.

night, and ostracises himself from his friends and family without cause or provocation.

Many men of versatile talents have been addicted to the abuse of alcohol. Alexander, Severus, Tasso, Coleridge and Gluck died of delirium tremens. Socrates, Seneca, Cato, Addison, Burns, Lamb and Poe were chronic alcoholics. One Italian scientist never accomplished anything when sober. Another of talents, a German poet, declared that only a genius could properly enjoy alcoholic beverages. Few people, however, will agree with this eccentric old German, as others besides men of genius appear to delight in the worship of Bacchus.

Great genius is incompatible with great happiness, says Lombroso, the Italian criminologist. The objectiveness of his exterior greatly antagonizes his intrinsic hyperæsthetic sensibilities; he is out of touch with his environment; he lives alone with his personality.

Genius, in common with insanity, is also characterized by marked precocity or its opposite extreme, youthful dullness, though rarely the latter. Many musical prodigies executed and composed before the age of ten, notably, Meyerbeer, Mendelssohn, Mozart and Beethoven. A large number of literary geniuses wrote works of merit previous to fifteen. We note Dante, Tasso, Goethe, Pope, Bryant and Byron among the poets, and Pascal, Bossuet, Comte, Voltaire, Hugo, Buckle and Fiske among prose writers. Linneus, Boccaccio, Volta, Burns, Balzac and Edison were dullards during youth. These lists might easily be quadrupled, as only the more noted and familiar names are mentioned by way of illustration.

A remarkable predisposition to functional neuroses and an especial frequency of the physical degenerative diatheses form another analogy between genius and insanity. Both are characterized by unstable or diseased higher nerve centres, upon which a definite neurosis is readily engrafted. Thus epilepsy, chorea, neurasthenia, paralysis in various degrees, rickets, phthisis, gout, rheumatism, Pott's disease, and other members of the neurotic and degenerate group occur with remarkable frequency among gifted men.

Though the genius be acutely sensible to extraneous influences, though his life be marked with predisposition to disease and premature decay, and flavored with a wealth of trouble and unhappiness, he is in like remark-



able for his long tenure of life. The extraordinary impetus he received as an heirloom at birth carries him on beyond the allotted time and thus insures the greatest good to posterity. The number of gifted minds who passed the ninety-year post is long and interesting. Xenophon, Cato, Michael Angelo, Von Moltke, and the elder Adams died at ninety. Chevreul 103, Titian 99, Hobbes 92, and Diogenes 91, all lived to pass ninety. The octogenerian list forms a brilliant array of talent, and is made up in part by such names as Voltaire, Hugo, Franklin, Tallyrand, Goethe, Kant, Newton and Holmes. The septuagenerian register contains scores of names of whom Galileo, Rossini, Emerson, Darwin and Locke are representatives.

Within the past two years an illustrious array of men of talent and genius have ended a career of studious research or general usefulness extending over three-fourths of a century. The obsequies of Billroth, Brown-Sequard, Charcot, De Lesseps, Holmes, Helmholtz, Parkman and Tyndall are still fresh in our memory.

Among those who died young, several illustrious examples appear, as will be seen by the following group, all of whom failed to reach the age of forty years: Mozart, Mendelssohn, Raphael, Burns, Keats, Poe and Byron. Literary men whose habits of life predispose to premature decay, artists and musicians make up the major part of this group.

Several of the most remarkable men of the nineteenth century are living to-day at an advanced age. We may mention Gladstone at 86, Leo XIII at 85, Bismarck at 80, Spencer at 76, Crispi at 75, Virchow at 74, Pasteur at 73, and Huxley at 72.\*

An indulgence in mysticism, symbolism, and things occult and æsthetic stimulate the fancy of the genius to productions, at times, of questionable utility or doubtful propriety. This characteristic is considered by Nordau<sup>25</sup> as symptomatic of degeneracy in its various forms, and our recent information respecting Osear Wilde's vagaries of moral sense seems to confirm this statement in part.

From our consideration of the predominant mental symptoms of genius we now turn to those mental manifestations which express a still greater degree of morbidity. We first come upon a class of cases called mat-

\* Died, June 29, 1895.

<sup>25</sup> Nordau. "Degeneration."

toids by Lombroso. The status of the mattoid bespeaks a connecting link between sanity and insanity. This group is made up of "gifted cranks," queer, odd, and eccentric geniuses, who, though actually insane, retain a high degree of intellectuality. As a rule these "mattoids" are hereditary neuropaths dominated by delusions of persecution, suspicion or grandeur, or imbued with ideas of universal reformation. The line of demarcation between the character of the mattoid and the variety of insanity most commonly found in the genius, namely, the so-called moral insanity, is necessarily indistinct and undefinable. The doubtful occurrence of a moral insanity as a definite mental disorder is now generally admitted, for in the majority of cases it is only a symptom of one of the involuntional or degenerative psychoses. Therefore, in a diagnosis of the predominant mental infirmity in men of genius, the term should be omitted and only the broader symptomatic classification be considered.

It is evident from what has already been said on the subject, that genius in many cases is on the threshold of insanity, and that only a slight increase in the instability of a given case would inevitably result in mental collapse.

Hammond<sup>27</sup> says that "The discrimination of the very highest flights of genius from insanity is a difficult and at times an impossible undertaking, for they may exist in one and the same person."

Nisbet<sup>28</sup> observes, "Genius and insanity are but different phases of a morbid susceptibility of or want of balance in the cerebro-spinal centres."

Diderot,<sup>29</sup> himself a degenerate, passionately exclaims, "Ah, how close the insane and genius touch! they are imprisoned or enchained, or statues are raised to their memory."

The fact that degenerative taint in a progenitor, originally produced by alcoholism, epilepsy, syphilis, insanity, or other morbid factors, may give rise to cases of insanity in one branch of the family and genius in another, and that whole families of degenerates comprised of talented, imbecile, and criminal members have often existed, shows the intimate relationship of the predisposing cause.

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<sup>27</sup> Treatise on Insanity.

<sup>28</sup> Insanity of Genius. London, 1891.

<sup>29</sup> Dictionaire Encyclopedique.

With the imperfect and wholly inadequate material at my command, I have endeavored to arrange the insane men of genius into several classified mental groups, thus attempting to diagnose the variety of the mental disorder manifested by each individual. Owing to the suppression, by the majority of biographers, of any allusion to a deteriorated mental state, and to the obscure description of the condition when too palpable to pass unnoticed, the results of an attempt at classification have not been more than moderately satisfactory. A few years' experience among the insane, has convinced me that a correct diagnosis of cases of mental disorder even by a personal examination is a problem of great uncertainty. It is, therefore, evident that the results obtained in attempting to classify the insane men of genius are still more unreliable, because the material upon which the diagnosis is based is derived from the biased writings of biographers and the opinions of the contemporary writers. Nevertheless, the attempt is made to divide the cases into the primary forms of mental disorder.

Comte, De Musset, Lamb, Lenau, Lucretius, Moliere,<sup>30</sup> Richelieu<sup>31</sup> and Shelley were subject to mania in one or other of its various forms.

Michael Angelo, Burns, Beethoven, Coleridge, Cromwell, Chopin, Cowper, Chatterton, Gray, Johnson, Lotz, Leonardi, Mill, Newton, Poe, Rossini, Raphael, Rousseau, Schiller, Schuman, Schopenhauer, and Tasso were victims of melancholia.

Baudelaire, Burke, Gogol, Linné, Pascal, Southey and Swift became demented in latter life, or suffered from paralytic dementia.

Pausanias, Bruno, Keats and Hoffman were cases of delusional insanity or paranoia.

Alfieri, Cæsar, Charles V., Flaubert, Handel, Mahomed, Moliere, Napoleon, Pascal, Petrarch, Peter the Great, Richelieu and Wellington were epileptics.

Biran's, Flaubert's, Johnson's and Renan's mental disorder is best expressed by the "*folie du doute*" of the French.

Amperé, Byron, Boyali, Brougham, Cardan, Descartes, Fodera, Gounod, Goethe, Hugo, Hastings, Haller, Mozart, Mendelssohn, Pope, Socrates and Ricci were more or less dominated throughout life by hallucinations

<sup>30</sup> Epileptic Mania.

or delusions, or else manifested other symptoms of mental vagary.

Many men of genius committed suicide. In some cases it was a sequel of mental disorder. The methods adopted are various, and almost cover the entire range of possible means of self destruction. Seneca and Lucan by bleeding; Lycurgus by starvation; Demosthenes, Hannibal, Lucretius and Chatterton<sup>31</sup> by poison; Cato, Brutus, Cassius and Marc Antony by sword or dagger; Blount, Balmaceda and Boulanger by shooting, and Nerval and Mainlander by hanging.

The following made one or more unsuccessful attempts at self-destruction: Clive, Cowper,<sup>31</sup> Cardan,<sup>31</sup> Chateaubriand, Fisher, Lessman Lamartine, Dupuytren, Rousseau and Schuman.<sup>31</sup>

Petrarch, Mozart, Peter the Great, Tacitus, Campbell, Donizetta, Volta, Hugo and Marcadante, were fathers of one or more idiotic or insane children, while fully seventy per cent. of geniuses have had insane relatives in their immediate family.

The relation of genius to criminality is as striking as its close approach to insanity, and more firmly cements the bond of union existing between this remarkable trio of degeneracy. A study of the genealogy of almost any degenerate genius discloses the fact, that the family has from time to time been represented in the criminal ranks. Marcus Aurelius, Pericles, Cardan,<sup>32</sup> Petrarch,<sup>32</sup> Luther, William Penn and Rembrandt<sup>32</sup> were fathers of one or more criminal children. The houses of Charles V., Frederick I. and Peter the Great, developed gifted, insane and criminal members. In a measure, these anomalies can be accounted for by consanguinity in marriage, lifelong dissipation and unusual stress of circumstances, yet there remains the faulty and degenerate ancestral basis upon which the above mentioned extraneous influences engrafted vice after vice until the lines became extinct.

From the mental we now turn to the physical points of dissimilarity between normal and abnormal man. Here the relationship to one another of the different members of the latter division is even more marked. These variations from the normal type have been observed from the earliest period in the authentic history of man. Homer portrays the villainy of Thersites, and

<sup>31</sup> Melancholia with suicidal impulses.

<sup>32</sup> Insane diatheses also present in family.

deems his picture incomplete without a description of his person. Pope's version thus:

"His figure such as might his soul proclaim,  
One eye was blinking and one leg was lame,  
His mountain shoulders half his breast o'erspread,  
Thin hair bestrewed his long misshapen head,  
Spleen to mankind his envious soul possest."

The physiognomy of the degenerate, as typified by the criminal, is highly characteristic. While the mental condition of the genius is closely allied to certain forms of insanity, his physical state more nearly approaches that of the criminal. The head presents the greatest deviations from the normal and is markedly asymmetrical. In the majority of cases, it is broad or brachycephalic with an increased cranial capacity. Extreme dolichocephaly is frequently observed, especially in the delinquent classes. Hydrocephalus was noted in Milton, Gibbon, Cuvier, Linnaeus and many others, while microcephaly characterized, Descartes, Dante, Foscolo, Gamba and Shelly. Cerebral anomalies of structure are found very frequently in men of genius, often analogous in position, extent and gravity to similar conditions in the brain of the instinctive criminal and degenerate insane. Thus occur meningeal thickenings, ventricular œdema, localized cerebral atrophy and softening, convolutional hypertrophy, and anomalies of the frontal and parietal convolutions and fissures.

In the majority of cases the hair of the scalp is long and abundant, while that of the beard is thin and scanty. The face is large and wide, the cheek bones prominent, the teeth irregular or crenated, and the lower jaw heavy, protruding and prognathic. The eyes are unusually far apart or close together, their individual qualities in the genius have formed themes for poets and biographers from time immemorial. The majestic brilliancy of the orbs of the Macedonian Alexander and Pompey the Great are said to have stirred the hearts of armies and to have filled the souls of their opponents with fear. Shelley's eyes were indicative of his wayward genius, his biographer describing them as "large and animated with a fiery dash of wildness." Carlyle's description of the eyes of Coleridge is well known.<sup>33</sup> "His deep eyes of light hazel hue, were as full of sorrows as of inspiration; confused pain looked mildly

<sup>33</sup> Carlyle. Life of Sterling.

from them as a kind of mild astonishment." The observant, cunning, steely eye of the criminal; the wild, haggard, roving eye of the maniac, and the expressionless eye of the idiot will be re-called in this connection while from the ophthalmologists we learn that strabismus, hemianopsia and numberless errors of refraction are very common among the degenerate classes.

The ears are misshapen and irregular; their lower lobe is elongated; they stand out from the head like wings, and the Darwinian tubercle is fairly constant in occurrence.

A very frequent abnormality is a prominent palatine ridge running from before backward, easily felt on the roof of the mouth.<sup>31</sup>

The shoulders in the majority of cases are stooped and rounded; lateral and angular spinal curvature is comparatively common, while both upper and lower limbs are long in proportion to length of waist. The feet are large and prehensile. The fingers long and tapering, with an exceptionally long or short index or little finger and a short, immobile thumb. Mancinism, or left-handedness, now regarded as a symptom of atavism or reversion of type, is often met with in the genius. Aristotle, Tiberus, Cato, Livy among the noted ancients, and Lamb, Michael Angelo, Raphael and Cardan among moderns serve as examples, and form a double quartette of illustrious mancini. Cutaneous anæsthesia, facial pallor and bodily emaciation, characterize the genius as well as the lowers members of the degenerate family. A variation from the family likeness or a non-resemblance to parents betokens the atavistic degenerate. Thus Cæsar, Napoleon, Voltaire, Humboldt and many others resembled neither father nor mother.

The universal physiological law asserting that excess in development of one part of the economy is counteracted by deficiency in another is well illustrated in the genius. In botany it is observed that plants which grow luxuriantly or attain an unusual size rarely flower. In the majority of cases great giants are imbeciles or have a low degree of mental power. Horace, Alexander, Aristotle, Plato, Epictetus and many others among the ancients were pigmies physically and giants mentally. Shortness of stature is also recorded of the

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<sup>31</sup> Dana. Medical Record, 1894.

majority of modern men of great talents. In this list we find Gibbon, Spinoza, Linnaeus, Moore, Campbell, Meissonnier, Mozart, Beethoven, Goldsmith, Heine, Balzac, Browning, Ibsen and scores of other names of equal note. A much more extended list is given by Lombroso (*loc cit*) who also names the following as being of tall stature: Goethe, Bismarck, Dumas, Washington, Voltaire, Johnson, Carlyle and Tennyson.

There appears to be a special group of American alienists, with a noticeable antipathy toward the Italian school, who attach slight significance to the subject of degeneracy as developed by criminal anthropologists, and incidentally aver that genius, criminality and their physical stigmata are purely accidental. They affirm that genius is no more a psychosis than blank ignorance, and that its concomitant neurosis is an accidental graft. Such statements entirely overstep the bounds of reason and relegate the labors of Maudsley, Galton, Lombroso, Benedikt, Nordau, Nisbet, Morel and the modern school of criminal anthropology to a very low degree of merit. They fail to properly distinguish between theory and fact, and betray an incredulity toward the results of modern scientific research that is, to say the least, very lamentable.

From the foregoing study it will be seen that genius, in the majority of cases, is a degenerative psychosis upon which is often engrafted a functional neurosis and a variety of physical stigmata which point towards a reversion of type. The difference between a degeneration and a neurosis is that the former is a reversion form and the latter a perversion of the normal type, or, in the words of Maudsley,<sup>30</sup> "Degeneration means, literally an unkinding, the undoing of a kind, or a change from a higher to a lower kind. That is, from a more complex to a less complex organism." In closing it only remains to be said, "That scholars and savants may by acquired knowledge, by the toilsome effort of study and research, make themselves a lofty position and gain the crown that science bestows upon her votaries. But the true genius is a born kind of an intellectual world invested by birth and nature with the royal prerogative." He is a magician whose wand sways all lesser intellects and whose innovations in science and art reach into the subtle powers of the sublime. Consciously

<sup>30</sup> Body and mind.

or unconsciously he is the natural enemy of conservatism, superstition and traditional methods; he is the apostle of reform and the advance agent of progress and civilization. To forestall and anticipate future events is the special prerogative of the genius. His master mind outlines some great and noble plan which the lesser intellects of future generations fill in, complete, and approve.

"This beaten highway is always open, and the difficulty is not to find those who will travel the old road, but those who will make a fresh one. Every age produces in abundance men of sagacity and considerable industry, who, while perfectly competent to increase the details of a science, are unable to extend its distant boundaries."<sup>36</sup>

Thus the genius is often unrequited during his life. He may build the foundations; alone and in silence, perhaps in obloquy, without sympathy or assistance, he lays the corner-stone. Contemporaries may ridicule; royalty may persecute; support may be entirely withheld, but the qualities which characterize true genius remain unaltered. Faithfully and wisely he draws the plan; labors throughout his long tenure of life; sacrifices pleasure and comfort to an insatiable ambition, and perchance death finds the foundation barely complete. Skilled architects of future generations rear the edifice and reap the reward, while the designer slumbers in dust, unrequited and forgotten. But there comes a time long years afterwards, when the selfish contentions of the builders have ceased and been forgotten; when a grateful posterity recognize the genius and merit of the original designer and fittingly reward his name and works which alone remain.

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# LEPRA ANESTHETICA AND SYRINGOMYELIA IN COLOMBIA.

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WHAT I shall refer to in the course of this article will naturally suggest the necessity of rendering ourselves, by a more careful study, capable of solving this question:—Are there not among 25,000 lepers, said to exist in Colombia, some who are not lepers at all, but only affected with an epidemic locomotor ataxia or syringomyelia; a disease which we know to ravage certain Indian tribes of British Colombia?

The pamphlet from which I extract, has been sent to me by Mr. McKinney, our minister in Bogota, Colombia, a gentleman who is deeply interested in the question of leprosy in that country.

Dr. Miguel Rueda Acosta in a clinical study of nervous leprosy of Colombia, after diagnosing nervous leprosy from muscular atrophies, myopathic, neuritic and myelopathic, speaks of a case of muscular atrophy which he examined in the Salpêtrière, in Dr. Charcot's service: it was a patient with infectious neuritis (1893). This case was very similar to *lepra anesthetica*. "The muscular atrophies, the forms of hands and feet were very much like those of lepers; but here the analogy stopped. There are no leprous antecedents, sensitive perturbations, spots, trophic lesions, such as ulcerations, osseous lesions, etc., nothing in the face that could suggest *lepra*."

"In short says Dr. Acosta, the diagnosis can be made in these cases, by any one knowing but a little about leprosy, and it can be made in a subject who has his neuritis developed in a country of endemic *lepra*, as the tropical regions, for instance. It would not be rare to find at the same time, spots of vitiligo and others so common in those regions. In these regions, before giving the diagnosis of *lepra*, it behooves us to think of the other neurites, which are not rare, especially of the neuritis consequent on paludism."

He goes on to say: there is a very difficult differential diagnosis; it is the diagnosis with syringomyelia, in which there are not only muscular atrophies like those of lepra, but also trophic lesions, and even analogous sensory disturbances. To make the diagnosis here, let us study the value and frequency of the symptoms which exist in leprosy, and not in syringomyelia.

*Spots.* "Usually the spots are an element of diagnosis, especially when they are accompanied with sensory disturbances. In tropical regions, the importance of this symptom is a little smaller, because the spots exist in many individuals, who are not lepers, and the spots of the lepers have not always distinctive characters. It is here that frequently the spots of the lepers are accompanied by sensory perturbances, which do not exist in the other productions, but this is not constant, and we shall see that in many cases mentioned by Zambaco, the distribution of two anesthetic zones, does not depend on the spots. On the contrary these are anesthetic because placed in a zone which is so. There are no clinical characters distinguishing the pigmented leper spots from pityriasis, for instance, although the latter has its parasite.

"The achromatic spots, when they are not anesthetic, cannot be distinguished from vitiligo, nor from the *Morphoeæ*. For many physicians the *Morphoeæ* are leprous spots. M. Zambaco tells us in this connection: 'Among the numerous lepers, whom I have observed in the Orient, affected with the anesthetic or tropho-neurotic variety, most did not present any pigmentation: so that it would be absolutely impossible to distinguish them from the syringomyelitic cases.

'I found achromatic spots in all lepers except two or three. I found them also in the healthy population of the Marqueses islands, in the proportion of 75%. I have not found any character whatsoever, which might help in distinguishing ordinary vitiligo from the vitiligo of lepers.'

"In subjects of the white race the spots are more important. For Marestang they are pathognomonic, but it must be borne in mind that it is not sufficient to be sure of the existence of the spots; the state of the sensibility must also be observed. The spots have twice been found in syringomyelia (Bruhl), in subjects of the white race. Let us remember that, according to Lucio and Poncet, and according to our clinical histories, the spots

are not constant in the kind of leprosy we are studying. In short, it may be said that the spots are a great element of lepra diagnosis, especially when they are anesthetic, but it is not possible to make a safe diagnosis with this element alone."

Dr. Gabriel J. Castaneda concludes, after study of the comparative sensitiveness, in syringomyelia and lepra anesthetica, that in both diseases there are sensory disorders and trophic lesions of various kinds, much alike; that the clinical elements of diagnosis may be found in the methodical study of the analgesic and thermo-anesthetic zones.

Perturbations of sensibility are found in the course of the nerves. In anesthetic leprosy, which is much like syringomyelia, the distribution of anesthesia is regular.

Dr. Quinquaud has carefully studied the subject of sensory disturbances in lepra, with the dynamometric aesthesiometer, and he is of the opinion that the elements of diagnosis in abnormal cases are to be looked for in the aspect of the sensory disturbances.

Dr. Acosta says that he is aware of the great importance which has been given to the dissociation of the sensibility for the diagnosis of syringomyelia.

Charcot in 1871, speaking of the same dissociation says, that "it is the starting point which distinguishes the syringomyelitic cause from the accidents, and without the presence of this symptom, which however, is not absolutely specific, all the clinic of the syringomyelia would have to go to the wall."

According to Acosta, the dissociation of sensibility may also be found in lepra, and he mentions a case of Thibierge of Marestang, and various cases of Zambaco, Rosenbach etc.

Anesthesia, according to Acosta, may not yet have appeared at the time of the examination; contraction may precede anesthesia. So says Poncet: according to him, there may be no anesthesia "even at the time when the fingers are gone."

Speaking now of trophic disturbances, it is an almost general belief, that in anesthetic lepra, all the trophic disorders of neuritis and syringomyelia may be met with. "It is especially in anesthetic leprosy," says Charcot, that we meet in all their development the lesions we have studied, in treating of the traumatisms of the nerves, with the exception of the zona, which I have found nowhere, we find in leprosy the whole series of

trophic lesions which we have described; a. the pemphigus (*P. leprosus*); b. Glossy skin; c. Muscular atrophy; d. Periostitis and neurosis."

Acosta says, "Scoliosis has not been indicated as a symptom of lepra, but it is not constant in syringomyelia either. We do not find in syringomyelia the spontaneous reabsorption of the phalanges, which has been indicated in almost all cases of the leprosy we study, but there are, on the other hand, the arthropathies which Charcot has compared to those of the tabetics."

Acosta concludes, "1. There are in tropical countries besides the cases of typical nervous lepra, other cases which manifest themselves principally by muscular atrophy, trophical lesions and deformations of the limbs.

"2. Before making the leprous diagnosis in these cases, we must bear in mind that myopathiæ, melopathiæ and neuritis present analogous symptoms.

"3. The diagnosis can be made and is perfectly established in myopathiæ, neuritis, and certain myelopathiæ, like the muscular atrophy type, Aran Duchenne, and the sclerosis lateral amyotrophy.

"4. The diagnostic with another myelopathia, the syringomyelia, may be made in clinic in the typical cases of both diseases.

"5. In anomalous cases of nervous lepra, the clinical diagnosis with syringomyelia is very difficult, and in certain cases the micro-biological study alone can decide.

"6. It will always be necessary to make a study of the distribution of the sensibility in the cases of lepra with instruments of precision, like the dynamometrical aesthesiometer, and it is thus that the clinical signs will be found to make the differential diagnosis."

# EVOLUTION OF THE NERVE CELLS.

By RAMON y CAJAL,

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Translated from the French by ADOLPH B. ROMÉRO and W. H. RILEY, M.D., Battle Creek (Mich ) Sanitarium.

THE work of His, who has succeeded in following in the embryos of mammals the different stages of development through which the ectodermic elements pass in order to transform themselves into nerve cells, have brought some important explanations to the interesting question of the evolution of the nerves cells.

Lachi, Cajal, Lenhossek and Retzius have confirmed the results obtained by His, and have continued this kind of research.

We will give here an abstract of this subject by utilizing the most recent data.

The epithelia of the medullary furrow of the ectoderm ( medullary plate) contains two kinds of elements; some long epithelial cells reaching from one surface of the membrane to the other, and some spherical cells, situated near the external face of this same membrane, which is grooved inward. These last corpuscles, called germinal cells, give origin by an active karyokinesis to the neuroblasts, or rudimentary nerve cells. The epithelia produces the cells of the ependyma and those of the neuroglia exclusively.

## GERMINAL CELLS AND NEUROBLASTS, AND THE DEVELOPMENT OF THE AXIS CYLINDER.

When the medullary furrow has closed in order to form the medullary tube, the inner surface of the embryonic marrow is thickened, and at the same time an important differentiation is produced in its cells.

Many of the germinal cells emigrate toward the peripheral half of the medullary wall. During their journey they are transformed into neuroblasts; that is to say,

into pyriform cells presenting an ovoid body directed either behind or within, and having only one relatively large expansion (process) which is extended lengthwise and passes toward the periphery, through the rudimentary gray substance. This primordial extension is the axis cylinder.

In preparations stained with carmine or hematoxylin, it is not possible to see the extremity of the axis cylinder in the course of its development; consequently His was not able to distinguish it in a very clear manner, and was therefore not prepared to successfully answer Hansen, who denied the growth of the nerve fibres toward the periphery; for, said he, no investigator has yet seen the end of the axis cylinder in the course of its development.

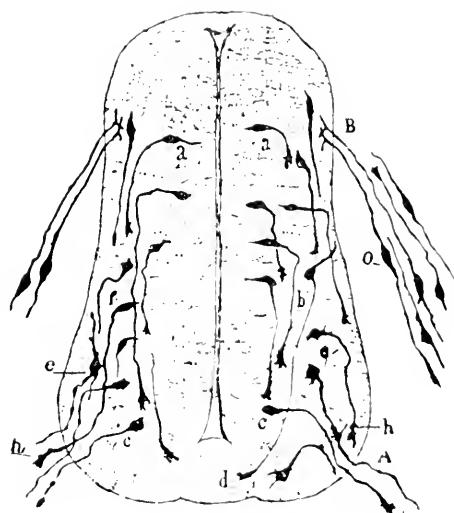


FIG. 1. Section of the spinal cord of a chick in the third day of incubation.

A. Anterior root; B. Posterior root; a. Very young neuroblasts; b. Other neuroblasts, more developed and probably commissural; c. Neuroblasts of the anterior roots; d. Cone of growth of a commissural axis cylinder, h and i. Cones of growth of anterior roots; e. Radicular cells which already present rudiments of protoplasmic branches; o. Ganglionic cells.

Our observations made on the spinal cord of a chick in the third day of incubation, have enabled us to solve definitely this problem and to pronounce ourselves in favor of

His and against the theory of Hansen. We have demonstrated that every primordial axis cylinder, while extending through the marrow, terminates by a special conic enlargement (*cone of growth*), the base of which, directed toward the side of the periphery, is furnished with numerous asperities and lamellary expansions which we may consider as rudimentary terminal arborizations. This cone of growth is like an amœboid mass, which, acting in the manner of a battering-ram, scatters the elements that are in its way and insinuates its lamellary expansions among them. Fig. 2 illustrates the neuroblasts as they appear in the spinal cord of a chick from the third or fourth day of incubation. There is seen also lying in the anterior region of the marrow, a group of cells which send their cones of growth to the anterior root; another group of neuroblasts disseminated through almost the entire gray substance, but accumulated more particularly in the most posterior region, directs its cones of growth toward the anterior commissure. Finally a small number of neuroblasts insinuate their nervous expansions in the peripheral region of the cord, the future territories of the white columns. Here these expansions form an elbow or divide themselves into a T, as the case may be, in order always to take a vertical direction. While such metamorphosis are taking place in the germinal cells the epithelium do not remain inactive.

#### EPITHELIUM CELLS AND THE FORMATION OF NEUROGLIA.

Every epithelial cell may be considered as made up of two parts; one part external and the other internal. The internal part is almost smooth, and, containing the nucleus, is separated from the expansions of the neighboring epithelium by some long spaces where are lodged a large number of neuroblasts (layer of the column of His); the external part is furnished with multiple short collateral expansions resembling thorns. These place themselves in contact with those that come from neighboring epithelial cells and thereby form a woof of spongy appearance, whose interstices will serve as a means of passage for the nerve fibres of the white substance (Marginal veil or Randschleier of His). According to His, these interstices are formed before the nerve fibres. Their role would therefore be very important, for they would then represent a kind of pre-estab-



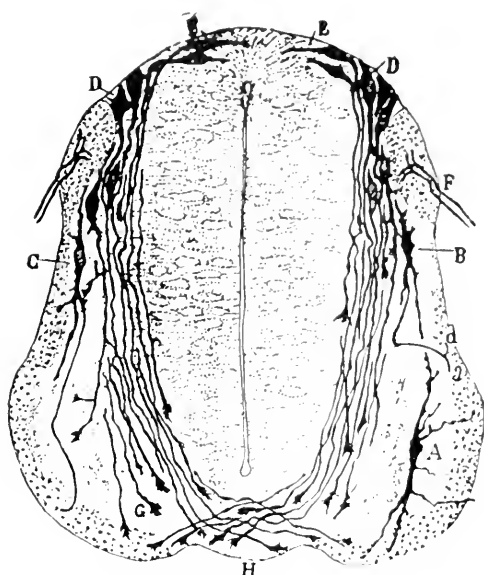


FIG. 2. Section of the dorsal marrow of an embryo of a chick in the fourth day of incubation.

A. Cell of the anterior root ; B. Cell of the antero-lateral column ; C. Cell of the anterior column ; D. Pyriform neuroblasts whose axis cylinder terminates at the level of the anterior commissure by a cone of growth ; E. Nervous primordial cells which still preserve the form of the spongioblasts ; F. Posterior root ; G. Cones of growth ; H. Embryonic anterior commissure.

lished, conducting canals through which the extremities of the axis cylinders would be obliged to pass. It appears to us, however, that the expansions of the epithelial cells, and consequently that the interstitial spaces of the marginal veil of *His*, are not formed before, but rather after the peripheral fibrillary substance ; we have seen in addition to this that in the retina and the brain all the most important differentiations of the nerve cells, the growth and distribution of the axis cylinders, take place at a time when the epithelial cells are still reduced to a state of simple radiating fibres deprived of all lateral expansions.

However it may be, we must consider the theory of *His*, who claims that the growth of the nervous fibres is always in the direction of the least resistance, and that their direction is determined by the pre-existence of the

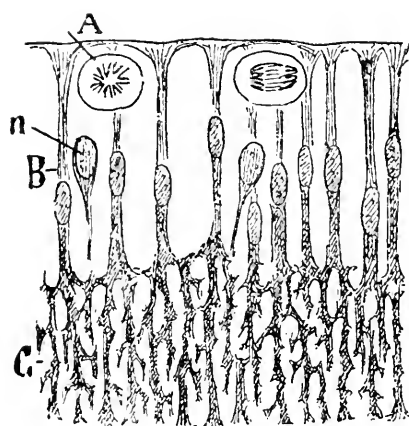


FIG. 3. Section of the embryonic spinal cord.

A. Germinal cells; B. Internal portion of the epithelial cells (layer of the pillars); C. External portion of the epithelial cells (reticular layers or marginal veil); N. Neuroblast (after His).

formation of neuroglia, as an ingenuous hypothesis and one worthy of study.

Perhaps it would be preferable to conceive in the neuroblasts a sort of positive *chemiotaxis* created by some nutritive substance elaborated in some other elements, either nervous, epithelial or muscular. One could also explain the power which incites the neuroblast to send its nervous prolongation toward a muscle, for instance by the existence of two electric states, different in the neuroblasts and in the muscle. This is the hypothesis recently brought forward by Strasser. Under the influence of a state electro-negative of the muscular fibre or any other cell which received the terminal arborizations of the nervous prolongation, there would be produced in the external pole of the neuroblast an electro-positive condition, which would induce it to work in the direction of the greatest difference of potential.

#### DEVELOPMENT OF THE PROTOPLASMIC EXPANSIONS AND OF THE COLLATERALS.

It is by the rapid method of Golgi that the last phases of development of the neuroblasts have been fully studied. The observations made on the spinal

cord of the chick by Lenhossek and us, independent one from the other, have completed the cycle of the works of His in showing in what way and at what time the protoplasmic arborizations and the collaterals of the nerve processes take their birth.

As soon as the axis cylinder is formed there may be seen on the neuroblast a short polar expansion, which might be considered as the beginning of a protoplasmic prolongation. Nevertheless this polar expansion is often absent, and the protoplasmic expansion usually commence by presenting themselves under the form of large excrescences of a thorny aspect, starting either from any point of the neuroblastic body or from the origin itself of the axis cylinder. Their free extremity is often provided with a varicosity.

The collaterals of the nerve process originate in their turn a few days after the formation of the protoplasmic expansions. They begin by a short appendix, departing at a right angle and terminating by means of a varicosity. The collaterals which appear first in the cord are those of the anterior column (from the fourth to the fifth day of incubation in the chick). In the following days one sees the beginning of the collaterals of the posterior and lateral columns. As to the bifurcations of the axis cylinders (fibres in the T or Y), we have already said that they begin to form during the period of growth of the primitive nerve expansion.

It is very easy to perceive the growth from life in the spinal cord of a chick from the fifth to the seventeenth day of incubation; that is to say, the march of the nerve fibre of the anterior root toward the muscles. Generally, the anterior root is completely formed by the fifth day, and its fibres have made such development that it is impossible to find their extremities within the limits of the anterior root. They are already found engaged in the neighboring muscular masses under the form of varicosities. Every axis cylinder during its journey gives off an extraordinary number of ramifications; thus in the embryo of a chick seventeen days old we have seen a large motor fibre producing more than thirty terminal branches, each one destined to a muscular fibre. The motor plates which are not yet formed are represented by a varicose fibre terminated by a small sphere placed on the muscular cell. In some plates the nerve fibre began to produce a certain number of short

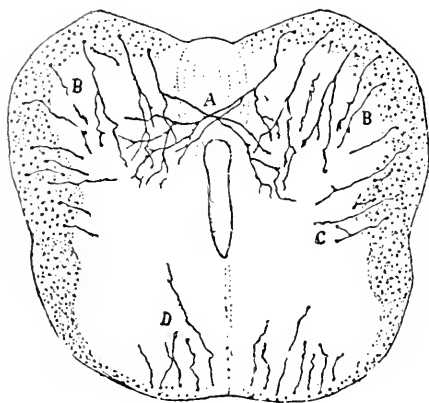


FIG. 4. Section of the marrow of the embryo of the chick of the seventh day of incubation.

A. Collateral fibrils of the anterior column, forming a crossing in the anterior commissure; B. Collateral fibrils, less developed, terminated by cones of growth; C. The youngest of the collaterals belonging to the lateral column; D. Collaterals of the posterior column.

excrescences, the first rudiments of the terminal arborization.

The preceding description is applicable to most nerve cells; however, there are a few which behave in a different manner and possess in their evolution peculiarities which may be expressed in a few words.

#### CELLS OF THE SPINAL GANGLIA.

In mammals, birds, batrachia and reptiles the nerve cells of the spinal ganglia are unipolar. It is only in fishes that one finds them in the bipolar form, similar to the olfactory cells of the nasal chambers, or to the cells of the spinal ganglion of the snail.

There seems to be an essential difference relative to the morphology of the ganglionic sensory cells in the higher vertebrates and fishes. But His has discovered a fact of the greatest interest. It is the following: the corpuscles of the sensory ganglia are bipolar in the embryos of mammals entirely the same as in adult fishes. This diminishes the distance that used to appear to separate them.

Of the two primitive expansions, the internal, gener-

ally more slender, grows along the posterior root and penetrates into the posterior column of the spinal cord, where, as our observations have been able to establish, it bifurcates in order to form an ascending and descending branch; we have been able to discover on this branch of these cells in the embryo of the chick of three days, a cone of growth directed toward the spinal cord; the external branch grows toward the periphery. It mixes with the motor fibres in the spinal nerve trunk and terminates finally in the skin, in the mucous membrane or in the musculo-tendonous organs of Golgi.

The bipolar cells undergo some modification during the growth of the spinal ganglia.

As has been very well figured by His, whose observations have been confirmed by us, on the embryos of some birds and reptiles, and also by Lenhossek, C. Sala, Retzius, E. Müller, Van Gehuchten, the extremities of protoplasm, which give root to two expansions, draw nearer one another, unite with each other gradually, and form on one side of the cell a pedicle of protoplasm, the only support for the two nerve fibres.

Fig. 5, which represents a spinal ganglion of the embryo of a chick on the fourteenth day of incubation, shows very distinctly all the transitions between the bipolar and unipolar phases. On examining this figure one receives the impression of a general emigration of the cellular corpuscles toward the periphery of the ganglion, the centre remaining reserved for the nerve fibres and the pedicle of bifurcation. These facts are then the proof that in the cellular ontogeny of the vertebrates there exist some evolutive transient phases which remain permanent in the fishes.

This is not all. Lenhossek has again demonstrated in certain fishes (embryos of the *Fristurus*) the presence of unipolar elements near the bipolar cells. It is thus seen that the transition from bipolarity into unipolarity begin in certain adult fishes.

It results from all these works that every sensory or sensorial cell is a bipolar corpuscle, or has been so in the embryonic epoch. All sensory or sensorial cells are always provided with two expansions: one peripheral, destined to receive the centripetal excitations produced at the surface of the body, and the other central, its office being to conduct to a nerve centre the excitations received by the first. Considered in this manner, the

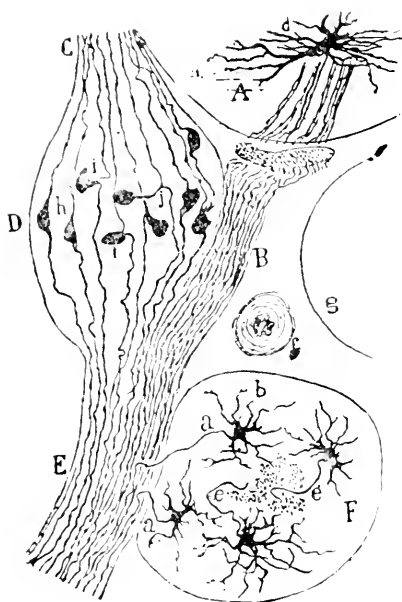


FIG. 5. Sympathetic and spinal ganglia of the cervical region of the embryo of a chick at the seventeenth day of incubation.

A. Spinal cord where is seen a radicular cell, d; B. Anterior or motor root; C. Posterior or sensory root; D. Spinal ganglion; E. Cervical spinal nerve; F. Sympathetic ganglion; a. Sympathetic axis cylinder forming the communicating rami and directed toward the corresponding spinal nerve; b. Protoplasmic expansion of the sympathetic cells; e. Sympathetic axis cylinders becoming vertical to form the longitudinal commissure of the ganglia; h. Fusiform cell of the spinal ganglion; i. Transition of the bipolar cell into the unipolar cell of the spinal ganglion; j. A distinctively unipolar cell; f. Artery cut across; g. Body of cervical vertebra.

sensory and sensorial cells form a well-defined variety, in which are naturally included:

(1) The cells of the spinal ganglia.

(2) The bipolar cells of the olfactory mucous membrane.

(3) The bipolar cells of the retina and the bipolar cells of the organ of hearing.

To this category belong also the fusiform corpuscles, discovered a short time ago by Von Lenhossek in the skin of the lumbricus, and whose existence has been confirmed by Retzius. These cells possess a large peripheral expansion terminating at the surface of the epi-

dermis, and a fine central expansion, which, penetrating into the interior of the body of the animal, goes to the ganglionic chain, where, like the fibres of the sensory root of the vertebrates, it bifurcates into an ascending and a descending branch. These singular corpuscles would represent, therefore, the cells of the spinal ganglia of the vertebrates, with the only difference that in worms, the protoplasmic mass of the cellular body (product of the ectodermic layer) has not retired into the interior of the body in order to form the ganglionic masses.

#### CELLS WITH PROTOPLASMIC TUFTS.

The cells of Purkinje of the cerebellum, the pyramidal cells of the brain, the ganglionic cells of the retina, pass also through the phase of the neuroblast. But the development of their protoplasmic expansions is made in a particular way and in an order which is important to know. That which appears at first after the axis cylinder is ordinarily the peripheral tuft, at first irregular and extremely varicose; later the protoplasmic peripheral boquet moves away from the cell, and thus is formed the vertical stem. The collateral branches of the stem and the basilar branches of the cellular body are always formed last. In the pyramidal cells of brain these basilar expansions persist and take even a large development. This is not the case with the cells of Purkinje and the ganglionic cells of the retina, where they appear almost at the same time with the expansion or principal stem. Here they are atrophied and disappear totally.

#### GRANULES OF THE CEREBELLUM.

The cells pass through extremely peculiar metamorphoses, which recall in part those of the unipolar corpuscles of the spinal ganglia. The cerebellum of newborn mammals, — mouse, rabbit, dog, man, etc., — possesses, as we know, above the molecular layer, a special zone formed by small corpuscles piled one against another, which are called superficial granules. But these corpuscles diminish in number in proportion as the cerebellum advances in its evolution; and there comes a time at maturity when they disappear completely. What is then the destiny of those cells whose

number diminishes precisely in proportion as those of the true granules develop? At the beginning of our study on the cerebellum we have already observed that the deep layers of the superficial granules are composed of long fusiform bipolar cells, extended horizontally and provided with long expansions of a nervous appearance, all directed parallel to the longitudinal axis of the lamellar cerebellus. It would have been thought that it was a question of parallel fibres similar to those which grow from the axis cylinder of the deep granules. Well, these bipolar cells are not in reality anything else but the primitive forms of the deep granules. By means of an emigration across the molecular layer subjacent, these corpuscles, contrary to the bipolar cells of the spinal ganglia, which from central become peripheral, take the direction of the lamellar of the cerebellum to assume there, all the characters of the adult granules. Before attaining that, these corpuscles pass through the following changes:

(1) From the body of the horizontal bipolar cell descends a protoplasmic appendix, which, little by little, carries backward the cellular body including the nucleus.

(2) This appendix which advances perpendicularly in the thickness of the molecular zone forms there an extended element, provided with two expansions, the ascending one extending itself to the superior part of the molecular layer by a parallel fibril; the descending one finishing freely near the zone of deep granules.

(3) When the cell carried away, so to speak, by and in the descending appendix, has reached the layer of the deep granules, one sees the ascending expansion becoming thinner and taking the appearance of an axis cylinder, which is continued by a parallel fibril; then the cellular body gives birth to three or several short appendices which do not take long in becoming the characteristic protoplasmic expansions of the deep granules.

This evolutive history, then, teaches us two interesting facts:

First, we see the parallel fibril fasten itself, so to speak, as a fixture and in a definite manner in the region which it occupied when at first it was represented by two extremities of the horizontal bipolar cell; this is exactly what we saw produced in the spinal ganglia: afterwards it is the ascending axis cylinder and the dig-



itiform expansions of the granule that are found to be the result of a protoplasmic expansion.

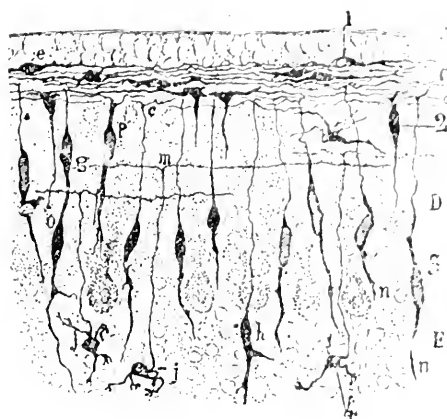


FIG. 6. Section of the cerebellum of a twelve-day-old mouse, wherein is seen the evolution of the granules.

A. Layer of the superficial granules; C. Layer of the horizontal bipolars; D. Molecular layer; E. Layer of the vertical or deep granules; first phase of the granule (horizontal bipolar cell; second phase of the granule (vertical bipolar by the production of the descending appendix which becomes the cellular body); third phase of the granule (the ascending axis cylinder is very perceptible and the inferior extremity of the cell already extends to the layer of the deep granules); fourth phase of the granule (completely developed granule).

#### SPONGIOBLASTS OF THE RETINA.

The nervous corpuseles which lack axis cylinder do not pass through the neuroblastic phase. Such is the case with the non nervous spongioblasts of the retina; that is to say, those which are not provided with an axis cylinder. In them the cellular body emits first through its inferior side a large bouquet of short varicose fibrils. This is nothing else than the first appearance of the terminal arborization. Later on the body extends itself in a vertical direction, and its inferior extremity is transformed into a stem to which is suspended the terminal arborization. Such is at least the evolution of a few spongioblasts of the retina in the chick and the rabbit.

The non-nervous spongioblasts of the retina are not the only elements existing with these evolutive and morphological characters in the animal series. They

are, as to their form, abundantly represented, and may be considered as a type of the phase of the morphological evolution to which come, for example, the cells of the invertebrates. Thus, according to the works of Bierdermann, Retzius and Lenhossek, the nerve cells of the ganglia in invertebrates (crustaceans and worms) are for the most part of unipolar form. Their unique expansion, in general very thick, furnishes then freely finished collaterals to the interior of the ganglia. When the cell is multipolar, all the expansions seem to have the same character; that is to say, they may all be considered as of a functional nature (Retzius). In the vertebrates the central cells on the contrary have experienced unimportant transformations which have brought them a long distance from the primitive neuroblastic form, so frequent in the centres of adult invertebrates. Some primitive forms, however, persist still, which have not suffered any differentiation in their expansions and resemble more or less neuroblasts. They are the spongioblasts of the retina, as we have just seen. The granules of the olfactory bulb. The sympathetic visceral cells and intestinal cells (plexus of Auerbach, of Meissner, etc.), and probably also certain cells of the cerebral cortex.

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**Vomiting of Nervous Origin.**—Ulinski (*Die Therapie der Gegenwart*, March, 1905), treated two hysterical patients for nervous vomiting according to the method of Winternitz. One was a case of gastric hyperæsthesia and the other was associated with genital reflex. The patients bodies were enveloped in a damp, cold sheet, upon which, over the region of the stomach, a coil of rubber tubing was placed. One end of the tube was conducted to a vessel on the floor and the other end led to another vessel filled with water at 40° R. and placed above the patient in such a manner that a running stream circulated through the coil of tubing. The patients were then wrapped in a dry sheet. The applications were made half an hour before administering food. The object of this procedure is to diminish the sensibility of the nerves of the gastric mucous membrane and thus cause cessation of the vomiting. Ulinski believes that the rubber coil exerts a direct sedative influence on the gastric nerves.

FREEMAN.

## THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, October 1, 1895.*

EDWARD D. FISHER, M.D., President.

### NEURITIS OF THE BRACHIAL PLEXUS, AS A SEQUEL OF PNEUMONIA.

Dr. W. M. LESSYNSKY presented a man, thirty-six years of age, who five months ago had suffered from an attack of neuritis involving the brachial plexus upon both sides. This condition had developed during convalescence from acute pneumonia. There was no history of traumatism or exposure to cold, nor was there any evidence of alcoholism, syphilis or rheumatism. He looked upon the pneumonic infection as the direct cause of the neuritis. The speaker said that at present there was a pronounced and typical paralysis of the left serratus magnus, and also some atrophy of the deltoid and supraspinatus. Upon the right side there was well-marked atrophy of the deltoid, with absolute anaesthesia in the cutaneous distribution of the circumflex nerve, and complete loss of faradic irritability. There was also atrophy of the supraspinatus.

## ATYPICAL HEREDITARY CEREBELLAR ATAXIA.

Dr. JOSEPH COLLINS presented a patient illustrating so-called hereditary cerebellar ataxia. The patient, a boy of eleven years, was the second of two children. Both his parents were still living, and aside from a marked tubercular history on the paternal side of the family, no hereditary taint could be traced. The child had never had spasms, and had passed through the ordinary infectious diseases of childhood. He began to talk and walk, and the teeth developed at the customary time. The mother said he had never been able to walk or talk like other children. He was always "stiff in his joints," "easy to fall;" etc.: he never climbed and played like other children, though he tried to do so. When four years old it was noticed that he was becoming very near-sighted, and glasses were applied, and had been worn since that time. In February, 1894, after returning from an outing with his father, he vomited a large quantity of liquid, and later, clotted blood. A few days before this he had had a fall down a flight of stairs, and to this fall the parents attributed his illness. He had been attending school since his fifth year, and had been repeatedly sent home on account of inability to make any advance. A year ago he was returned from school permanently. For a year or more there had been periods when his mother said he could not use the left side of the body, and at such times, the arm and leg had become very unsteady, and his speech unintelligible. Great difficulty had been experienced in keeping him warm, and the body surface of good color in the winter. The speaker said that mentally the boy was peevish, irritable and irascible. He was affectionate, fond of animals and pictures, and while he could be led, he could not be dominated. When seen on July 21, 1895, it was noted that he was tall for his years; face looked very old; the skin looked and felt dry and the flesh pul-taceous; no enlargement of the thyroid gland; right side of face more innervated than left; very slight grip in hands; knee jerks exaggerated, especially on the right; elbow-jerk lively; slight ankle clonus; gait

shambling and reeling; stands fairly well with feet wide apart; genital organs extremely undeveloped. His manner of rising from the recumbent posture resembled that of one suffering from progressive muscular dystrophy, except that he did not "crawl up the legs." There was no nystagmus, but the fixation power of the eyes was not good, and there was slight weakness of the external ocular muscles. There was progressive myopia, but Dr. W. A. Holden who examined the eyes, stated that the background seemed normal. His speech was ataxic and thick, and sometimes quite jerky. It was almost impossible for him to go up and down stairs. Mentally he was very defective.

The speaker said that this case would tend to corroborate the view taken of these cases by Nonne, to whom more than to any one else credit was due for calling intelligent attention to them. Marie who referred to Nonne's cases, and a few which he had observed himself, as cases of heredo ataxie cerebelleuse, took for granted a factor which apparently was not always constant or necessary, viz., heredity. The sister of this boy who died in her second year was affected in the same way, for the parents gave a very clear description of her condition. That it is familiar is universally admitted. Another element claimed by Marie to be constant was optic atrophy, yet in some cases recently reported, this symptom was absent. The condition has been more commonly observed in patients older than the one just presented. It was hardly necessary to refer to the apparent relationship between Friedrich's disease and this condition.

## A CYSTIC TUMOR.

Dr. L. STIEGLITZ presented a young woman whom he had exhibited to the Society in January, 1893, after she had been operated upon in June, 1892, for a cystic tumor of the brain, situated in the right arm centre (*American Journal Medical Sciences*, 1893).

The cystic tumor was sub cortical, about one inch and a half deep, and its walls had been found perfectly smooth. About an ounce of straw-colored serous fluid had escaped from the cyst at the operation. In discussing the case at the time, it was clearly shown that the cyst could not have been of vascular, nor of parasitic origin, and the conclusion had been arrived at, that a glioma was at the bottom of the trouble, and on that account, a recurrence of the growth had been feared. During the following two years the patient had done very well, and had recovered the use of her arm sufficiently to attend to her housework, but had been subject to more or less frequent focal epileptic seizures involving the left hand, or the entire left arm and side of the face. During this period she had also had about five general convulsions which had set in with the same focal symptoms. In March of this year, the patient had begun to complain of headaches, the power in her left hand had again perceptibly diminished, and the focal seizures had occurred more frequently than ever—from one to three times a day. Dr. Koller, who had examined her eyes frequently, found a beginning cloudiness and swelling of the disc for the first time. A recurrence of the original neoplasm, or a re-filling of the old cyst was suspected, and Dr. Gerster performed a second operation upon the patient in April of this year. Upon exposure of the brain, a very small maroon-colored mass about the size of a filbert was found at the point where the cyst had been. This mass was removed, and as it evidently infiltrated the surrounding brain tissue, a broad ring of the latter was excised. The patient made a good recovery. She had two severe general convulsions when the dressings were first removed, but had been free from all seizures ever since—a period of six months. Her arm was at first completely paralyzed, but she had

now recovered its use to a certain extent, the only marked paralysis left pertaining to the index finger, which could not be fully extended, and to the thumb, which could be flexed, extended, abducted and adducted only very imperfectly. Tactile sensibility was less acute in the paralyzed limb than on the other side. There was a very marked disturbance of the muscular sense, the patient being very uncertain about passive movements in her finger joints, especially in those of the thumb and index finger. The deep reflexes of the arm and the knee-jerk were greatly exaggerated on the affected side. Curiously enough, they were also distinctly exaggerated on the healthy side, though to a far less degree. The microscopic examination of the excised tumor was very interesting. The old cyst wall was found collapsed; originating from a point in this wall a small spindle-cell sarcoma was found. This could be seen in the specimen. Sections through the ring of brain tissue removed, showed the new growth to be of a very malignant and complex type. From its character at different points it would have to be termed a melano-glio-angio-spindle-cell-fibro-sarcoma. The brain tissue was infiltrated, and no doubt sooner or later the patient would have a recurrence of the growth.

The case showed that cystic tumors of the brain were not so innocent in character as many would believe, and on that account the excision of the cyst wall would be advisable in all cases of the kind. If this were not possible at the first operation, it could be easily done at a subsequent operation, a few months later when the wall would be found compressed into a small, solid mass.

#### DISCUSSION.

Dr. B. SACHS expressed the belief that the original cyst might very well have been of hæmorrhagic origin.

The PRESIDENT said that the clinical history of the case prior to the first operation would throw light upon that question.

Dr. STRIEGLITZ said that in order to save time he had omitted the details of the original history. The patient had been perfectly well up to December, 1891, when she had been suddenly taken with twitching in her right thumb and forefinger. The twitching had spread rapidly up the arm to the face, and had ended in a general convulsion. In December, the patient had had

three or four more general convulsions of the same character, but none after this time. She had been subject, however, to frequent focal seizures confined to the right hand. When first seen in December, there had been no loss of power in the arms. The weakness in the arm had developed the following March, and had gradually increased, until in June, the time of the first operation, it had become very marked. In spite of the absence of all general cerebral symptoms, the diagnosis of progressive, destructive focal process, probably a tumor, had been made. The slow and late development of the paralysis after the onset of the symptoms of cortical irritation positively excluded any vascular lesion.



## AMYOTROPHIC LATERAL SCLEROSIS.

Dr. WILLIAM HIRSCH presented a case of amyotrophic lateral sclerosis which had developed in a patient who had formerly had anterior poliomyelitis. M. D., forty-five years of age, gave a negative history as regards syphilis and heredity. About three years ago he noticed a stiffness and weakness in the left leg, which gradually became worse. After some time, the right leg also became affected. He soon became unable to use his legs in working his sewing machine (being a tailor), and his gait became so much impaired that he was not able to stand or walk for any length of time. When he came under treatment, four weeks ago, he had complained of nothing else, and there were no sensory symptoms or disturbance of bladder or rectum. The examination showed an atrophy of some of the muscles of the trunk and the upper extremities. The right deltoid, the supraspinatus, and especially the thenar eminence on the right hand showed a fairly marked atrophy. There was fibrillation in the muscles of the trunk and shoulder, increased by tapping them with the percussion hammer. There was also diminished electric excitability, and in some muscles, the reaction of degeneration was present. There was no atrophy in the muscles of the lower extremities, and the electric reaction was perfectly normal in these parts. The tendon reflexes of the lower extremities were considerably increased, the knee-jerks were extremely exaggerated, and there was bilateral ankle clonus. The last very frequently came on spontaneously to the great annoyance of the patient. Sensation was perfectly normal in all parts of the body, and the senses of pain and temperature were unimpaired. There were no abnormal conditions in the function of the cranial nerves, with the exception that the tongue was slightly atrophied, and there was a marked masseter reflex. Speech was in no way affected. There was no nystagmus, and the ophthalmoscopic examination showed perfectly normal conditions.

This was evidently a typical case of amyotrophic lateral sclerosis, and the man was brought here because of a possible relation between the present disease and an old affection.

An examination of the case at present showed a very marked atrophy of the muscles of the left shoulder and upper arm, but the patient did not think it worthy of mention, as he said it had existed as long as he could remember. This appearance, together with the history show that he had had in childhood an attack of infantile paralysis. This brought up the interesting question: Does there exist any relation between the present disease and the anterior poliomyelitis, and if so, what is the nature of this relation? Dr. Hirsch said that Ballet and Dutil were the first to enter upon a discussion of this subject. They pointed out that an infantile spinal paralysis might give rise in later life to various affections of the spinal cord, but that they were all limited to the gray matter of the anterior horns. The irritation of the cord by the old lesion enfeebled, they said, the medulla and made it a *locus minoris resistentie*, which on any occasion might become subject to further disease. Charcot expressed the view, that there existed in some individuals a certain disposition, a kind of hereditary vulnerability of the ganglion cells of the anterior horns, which at one period of life might give rise to an acute poliomyelitis anterior, and at another, to a progressive muscular atrophy, so that in fact both diseases would form different periods of one and the same pathological process. In some cases of infantile spinal paralysis, Charcot said, the old scar which was produced by the inflammatory process in the gray matter of the anterior horns, formed a latent, but permanent, inflammatory focus, which at any time might light up afresh, and cause a new set of symptoms. A similar view, the speaker said, was held by Strümpell who, on the theory of the infectious origin of poliomyelitis anterior, compared the scar in the anterior horns to an old tubercular focus which was capable of setting up a new inflammation at any time. So far as he knew, the case differed from all others recorded in literature in that the later disease was not limited to the anterior horns of the gray substance, but that other parts of the cord were also involved, *i. e.*, both lateral pyramidal tracts. He would not attempt to decide which of the theories applied to this case, but it could be proved with absolute certainty by the history of the clinical symptoms that the pathological process of the present diseases started from the place where the old scar of the poliomyelitis anterior was located. This lesion lay apparently in the

left horn of the cervical region of the cord. As the man first noticed weakness and stiffness of the left leg, the process must have approached first the left pyramidal tract which lay next to the scar. Then, after the affection of the right horn, which showed itself in atrophy of the muscles of the right upper extremity, the pathological process spread over to the right pyramidal tract, causing a spastic condition in the right leg. Whatever the theory regarding the nature of the relation between the two diseases, there could be no doubt that there existed such a relation between infantile spinal paralysis and various spinal diseases of later life. Further observation and study of similar cases might throw more light upon this subject, as well as upon the etiology and nature of the diseases in question.

## A REPORT OF A SERIES OF CASES OF MULTIPLE NEURITIS IN INFANTS IN THE CITY OF BRIDGEPORT, CONN.

Dr. GRÆME M. HAMMOND read a paper in which he reported a series of cases of multiple neuritis in infants in the city of Bridgeport, Conn. There were ten cases in all, ranging from four and a half months to four and a half years in age. In all but two cases there were distinct premonitory symptoms of headache, vomiting and fever. Following this there was a gradual and progressive paralysis, usually beginning in the feet, and extending to the upper extremities, and in some instances, involving the muscles of the trunk, and in two cases, the muscles of deglutition. Accompanying the paralysis there was pain, both spontaneous and on passive motion, and the nerve trunks were very sensitive to pressure. The area of pain and tenderness accompanied the extension of the paralysis. The reflexes were abolished, and the electrical reactions of degeneration were well marked. Two of the cases exhibited symptoms of spinal meningitis, in addition to the neuritis. One case, the youngest, four and a half months old, died after an illness lasting one month. The muscles of deglutition became paralyzed, and the child died from exhaustion. In the other cases, recovery began in from four to six weeks, but was not completed until from three to four months. No similar cases were discovered in neighboring towns. After referring to the epidemics of anterior poliomyelitis which have in the past been reported, both in Europe and in America, the reader concluded by accepting the theory of their microbic origin, and expressed his belief that these cases of neuritis were due to a similar cause.

The author expressed his indebtedness to Dr. John C. Lynch, Dr. Samuel M. Garlick and Dr. J. W. Wright, of Bridgeport, for the reports of cases, and for their courtesy in affording him the opportunities for personal examinations.

## Asylum Notes.

BY R. M. PHELPS, M.D.,

Rochester, Minn.

*A Synopsis of One Year's Admissions.* It has been frequently asserted, and about as frequently admitted, that our statistical reports concerning the insane are very unreliable. We have ourselves frequently asserted that the data were necessarily very imperfect. Many a patient comes to us, with none or a trivial cause assigned, with a duration assigned which is usually too short, and with a slight and erroneous account of the beginning stages and of family tendencies. And as we search the literature, few are the attempts which we find to unsnarl this tangle, a task which can only be done approximately by the patient, laborious effort of one having a personal knowledge of each case. Dr. Christian, of Pontiac, Michigan, records a worthy effort to obtain the true mental state of those recovered by studying the future history of those so sent away. This method requires years of waiting, and the almost impossible task of keeping track of scattered patients.

The present is a brief attempt to ascertain the standing of the insane patients admitted to the Rochester State Hospital during the year from August 1, 1895, to August 1, 1896, from the standpoint of the present date, November 1, 1896; an attempt to ruthlessly judge every case, ignoring every previous judgment, and striving to not swell the recovered list one bit more than it deserves. We first present a tabulated statement of the results and afterward give a few explanatory statements.

### AGES OF THOSE ADMITTED.

	MEN.	WOM.		MEN.	WOM.
80 to 90	0	4	40 to 50	24	23
70 to 80	4	4	30 to 40	38	28
60 to 70	19	9	20 to 30	13	10
50 to 60	17	13	10 to 20	6	5
	—	—		—	—
Totals,	40	30	Totals,	111	95

## DURATION OF INSANITY.

Under 6 months,	24	10	2 to 4 years,	37	28
1 to 6 months,	10	35	4 to 6 years,	18	13
6 months to 1 year,	17	5	6 to 10 years,	10	4
1 year to 2 years,	22	14	Over 10 years,	13	15
Totals,	73	54	Totals,	78	60

	MEN.		WOMEN.	
Total No. of patients considered.....	150		124	
No. died.....	19		14	
No. of these over 50.....	11		5	
No. over 50 died of general paresis.....		1		0
No. died of exhaustion, senile.....		7		4
No. died exhaustion with acute psychosis.		0		1
Pneumonia, heart and kidney disease.....		3		0
No under 50.....		8		9
No. under 50 died of consumption.....		3		4
No. under 50 died of general paresis.....		3		0
No. died acute delirious mania.....		1		2
Exhaustion of imbecility.....		0		1
Typhoid fever.....		0		1
Exhaustion in acute psychoses.....		1		1
No. recovered.....	18		21	
Left for home.....		16		20
Still in the institution.....		2		1
Those of above having previous attacks...		7		9
Those named recurrent.....		0		3
Duration of attack over 6 months.....		0		0
No. strongly improved.....	20		16	
No. of these at home.....		14		13
No. still here.....		6		3
No. still unimproved or mildly improved....	93		73	
Those strongly defective.....	26		15	
No. imbeciles.....		5		3
No. epileptics.....		8		2
No. strongly paranoiac.....		9		9
Adult hereditary chorea.....		0		1
General paresis.....		4		0
The remainder unimproved.....	67		58	
Of these those over 50.....	16		15	
Age 80 to 90.....		0		3
" 70 to 80.....		3		4
" 60 to 70.....		7		4
" 50 to 60.....		6		4
Character of these cases over 50. Show-				
ing senile or organic change.....		9		12
Senile melancholia.....		2		1
Long demented.....		1		2
Mild mania of long duration.....		2		0
Syphilitic and alcoholic.....		1		0
Severe acute mania, previous eccentricity.		1		0

Of those under 50 years of age.....	51	43	—
Ages 40 to 50.....		7	14
“ 30 to 40.....		23	9
“ 20 to 30.....		20	19
“ 10 to 20.....		1	1
Character of those under 50 years.....	51 51	43 43	
Duration of insanity 2 to 17 years.....		17	17
Alcoholic dementia.....		4	1
Previous attack at least 2 years before.....		2	1
Strong evidence of being defectives.....		10	5
Less strong evidence of defectiveness.....		6	4
Remainder still unexcused.....	12	12	
No. having fairly acute attack.....		7	10
No. admitted heredity.....		4	5
No. parents first cousins.....		1	0
Total.....		150	124

It will be noted in the above tables, that by using in their order the most powerful reasons of non-recovery, we cut off one detachment after another from the complete list of unimproved. The explanation concerning the deaths is sufficient. Cases of general paresis or syphilitic brain disease are not expected to recover. Nor will senile dementia, whether of the quiet decline or the more atheromatous and rapidly progressive order. Nor will, of course, epileptics, imbeciles or the hereditary choreas. Nor will the strongly paranoiac be expected to recover; nor, except in occasional cases, those over fifty years of age; nor will those long demented or the alcoholic dementias; nor will senile melancholias, usually of strong senile trend; nor, beside exceptional cases, will those of over two years' duration. The judgments upon these classes will be quite uniform in all probability. A few comments will be in place. Those designated as "imbeciles" were not all positively so stated, yet the facts quite justify us, even if occasionally they are a few grades above the ordinary imbecile case. Then the class, ten men and eight women, designated as "defective" yet not fully imbecile, and the class, six men and four women, still a few grades nearer the normal, viewed one by one, are each of them usually young, presenting usually no acute attack (though occasionally exacerbations of excitement or of depression), and only the dull low level of intelligence and ability, reaching back indefinitely, and supposedly through life. Many of them have, of course, grown somewhat more defi-

nately troublesome of late, determining their commitment.

Finally having disposed of the most powerful causes determining against improvement or recovery, we find only ten men and twelve women, for whom we shall offer no special excuse.

"Defectiveness" is undoubtedly the most important element brought out by the analysis. A typical defective is a few grades above the feeble-minded, and must include mental and moral perversions, as well as the more pure feebleness of mind. Often there is considerable brightness, usually there is no acute attack. It grows insidiously. Of such is the character of the sixteen and twelve designated, reaching up, of course, many of them into the adolescent type of insanity.

Of cases having a like character, beside these sixteen and twelve, we may add the imbeciles, five and three, the epileptics, eight and two, while of the paranoiacs, seven and five, are surely defective, and among the improved, recovered and died, we may select thirteen and eleven more as having strong alliances to this class.

The powerful central and strong impression then is of a heritage so powerful as to impress itself gradually and insidiously during the adolescent period. Combining these with the senile cases, we have approximately 108 out of the 274 cases. Not that the defectives do not have their recoveries; they occasionally recover quite well. Indeed, a paroxysmal "up-and-down" motion is a characteristic of some forms. The general tendency is, however, to a gradual slow decline. We have discarded in our above list all ordinary and very temporary recoveries.

Of course, defectiveness, if the finer analysis were pushed, might be made to include about all of insanity. It is not, however, in this extreme sense that we mean it.

Where do the typical acute psychoses have their place then? those of functional character, without strong defective or degenerative base? They tend evidently to come in middle life. Acute delirious mania seems to strongly prefer people of the ages from thirty to fifty. Ordinary typical acute mania, shows also a mild preference in that way, only one of our cases being over fifty years of age, and six being below thirty. Acute melancholia shows less marked preference, senile melancholia being somewhat frequent, and a good many cases being below thirty years of age.



Of "periodicity" so strong as to deserve the name "recurrent," we note one case as remarkable. A fine-looking and intelligent old lady, now sixty-five, who has been the mother of several children, had a first attack at twenty years of age. Though somewhat irregularly, she has had them ever since, occasionally severe, frequently slight. Her maniacal attacks, though quite severe at times, are usually short, being over in a week or two. Her quiet spells often last six months. At her best she is still quite clear and shrewd, though a little of mental weakness is to be noted. Her mother had a similar trouble.

Our population coming from a State which admits old and young, rich and poor, and completely without cost to themselves, and which has no private asylum within its bounds, our conclusions ought to be the more accurate. Our recovery rate will, of course, be somewhat increased by subsequent recoveries.

***Pathological Supplement to the Annual Report of the Government Hospital for the Insane, 1893.***—I. W. Blackburn, special pathologist.

#### SUMMARY.

The one hundred and four cases of melancholia studied in this report comprise all the examinations of this form of mental disease made since the establishment of the pathological department of the hospital. Ninety-four of the cases were males, ten females; eight of the males were colored; all of the females were white.

The clinical subdivisions of melancholia have not been given, and the division of the cases into acute and chronic has only been indicated by the duration given in each case. It must be understood that some degree of terminal dementia existed in all the chronic cases, and that in some the mental failure was almost complete. In some of the cases senile failure of mental power must be assumed to have existed in addition to the symptoms of melancholia.

The study of these cases was undertaken without the hope of adding materially to our knowledge of the special pathology of melancholia, but rather with

especial reference to accompanying physical conditions, many of which may have had a direct causative relation to the disturbance of brain function.

It was at first intended to group the cases in a tabular form, but it was soon found that almost every case possessed some individual interest; enough, it was thought, to justify a brief synopsis of each and a summary of the most interesting points observed in all.

Lesions of the skull of pathological importance were not very common; exostoses on the inner table were found in two cases; the skull was thinner than the average in twenty cases, one of which was colored; it was thicker than usual in twenty-seven cases, five of which were colored; in seven of these the skull was denser than normal. Abnormal density without appreciable thickening was observed in eleven cases. In many of the above the deviation from the average was very slight and unimportant and probably had nothing to do with the mental disease. Slight peculiarities of shape were observed in a few cases.

The dura mater was adherent to the skull bones in many cases, but principally in elderly persons, and, therefore, of slight pathological value; various degrees of internal pachymeningitis were found in twelve cases and in one of these a large hæmatoma had been formed. Hemorrhage into the subdural space was present in two cases; in each the ruptured vessel was in the pia mater.

The pia mater showed more or less opacity and thickening in many cases, and in all showing atrophy of the convolutions; the membranes were œdematous. In thirty of the cases the pial veins were noticeably full of blood; in the remainder of the brains examined they were normal in appearance or comparatively empty. Forty-three cases showed some disease of the cerebral arteries, though in the greater number it was slight.

In almost every case where the disease had existed for some time decided atrophic changes were found in the convolutions, nearly always most noticeable over the fronto-parietal convexity and the anterior portions of the median surfaces. Some cases of remarkable shrinkage were found in those of less than a year's duration, and some very heavy brains showed some atrophy of the convolutions over the anterior portions of the hemispheres. Of course, the atrophy due to miasmatic conditions and to old age must be taken into consideration in individual cases, but the frequency of the localization

of the shrinkage in the supposed mental regions suggests a direct relation of the atrophy to the mental failure.

The average weight of the male brain which were weighed, 88 in number, was 47.005 ounces; of the female brains, 7 in number, the average was 43.62 ounces. The heaviest brain of the males weighed 61 ounces; the lightest weighed 35 $\frac{3}{4}$  ounces. The heaviest female brain weighed 48 $\frac{1}{2}$  ounces; the lightest, 40 $\frac{3}{4}$  ounces. The average weight of the brain in 49 males over 50 years of age was 46.418 ounces; in 39 males under 50 years of age the average was 47.743 ounces, showing that the age of the patients had something to do with the reduction in weight. In 13 cases of males in whom the mental disease had existed less than a year the average weight of the brain was 49.1 ounces. It should be noticed, however, that four of these brains weighed over 50 ounces, and one weighed 61 ounces. The average weight of the brain in 75 males in whom the disease had existed over one year was 46.64 ounces.

Chronic softenings of the brain were found in twenty-six cases; in the majority, however, the lesions were small. They were usually located in the cortex or in the region of the basal ganglia, and were commonly associated with atheroma of the cerebral arteries.

General reduction of consistence, œdema, and enlargement of the perivascular spaces were found in various degrees in nearly all the chronic cases, and frequently in minor degrees in the more acute cases. Dilatation of the ventricles was commonly found in connection with œdema and atrophic changes in other parts of the brain; the ependyma was granulated in a few cases.

Lesions of the cerebellum, pons, and medulla were not common, and the spinal cords were generally found to be normal.

The microscopical examination of the brain added but little to the naked eye appearances. In the chronic cases degenerative changes in the nerve cells were demonstrated in nearly every case, but it is, of course, difficult to decide how much of this change should be considered as directly related to the mental disease either as cause or effect. Vascular degenerative changes were common, such as tortuosity of the vessels, pigment granules, and red blood corpuscles along the vessel walls, etc. In the acute cases it must be acknowledged that structural alterations could not usually be discov-

ered, though some showed decided degenerative changes in the nerve cells and vessels, which were, no doubt, closely connected with the mental symptoms.

Of the diseases of other organs, tuberculosis of the lungs was found in thirty-five cases. The disease was found accompanied by tubercular infection of other organs, and was in many cases the direct or indirect cause of death. Three deaths were due to pneumonia; one to gangrene of the lungs, and two to tubercular pyothorax.

The heart showed valvular disease accompanied by hypertrophy and dilatation in at least eight cases, and chronic disease of the valves without important effect upon the heart walls occurred in at least fourteen others. There were six cases of atrophy of the heart, usually in connection with phthisis, one case of specific myocarditis with perforation of the septum, and one aneurism of the aorta.

The diseases of the kidneys were usually chronic in character. In thirty-five cases they presented a degree of contraction deemed of pathological importance, shown by adhesion of capsules, granular surfaces, cortical atrophy, and reduction in weight. In sixteen cases lesser degrees of contraction existed, but the function of the organs had probably been very slightly impaired. There were four cases of pyelonephritis with cystitis; two of tubercular abscesses of the kidneys, one horseshoe kidney and one kidney with two ureters.

As time did not admit of microscopical examination of all the kidneys it is probable that some cases of acute degenerative changes in the renal epithelium escaped observation, but as these are usually dependent upon other physical conditions and could have had no relation to the mental disease the omission was considered unimportant.

Gross lesions of the liver were uncommon. Passive congestion was of frequent occurrence in connection with heart and lung diseases; tubercles were found in a few cases and biliary calculi were found in thirteen cases.

Five malignant tumors were found, all of which were carcinomata; in one case the prostate gland was the seat of the primary tumor.

By FRANK A. NORBURY, M.D.,

Jacksonville, Ills.

*A new Journal that ought to be good.* We are in receipt of the announcement that in January, 1896, the first number of the *State Hospitals Bulletin* will appear. It purports to be a quarterly report of clinical and pathological work in the State hospitals for the insane and their pathological institute, and is published by the authority of the State Commission in Lunacy. Editors ex-officio, The President of the State Commission in Lunacy, The Medical Superintendents of the State Hospitals, and the Director of the Pathological Institute.

The editorial committee are: P. M. Wise, M.D., C. W. Pilgrim, M.D., S. H. Talbot, M.D. Collaborators are the assistant physicians and medical internes of the several State hospitals. State Hospital Print, Utica, New York.

About six months ago no doubt most of our readers examined the St. Lawrence Hospital Report and were struck by the marked improvement it showed in the clinical and pathological features over former so-called asylum reports in which we dived in vain.

We have no doubt that this impetus is now manifesting itself in a more decided advance. The State Commission desire to concentrate and chrystalize the work in a scientific way in all the hospitals under their care. We have heard many rumors as to what to expect, and this announcement is an earnest step in a right direction. We will only be too glad to aid this project and we will carefully cull from this report all that is good for the benefit of our readers, and that they will be valuable cannot be doubted. We can prophesy that with the appearance of these scientific bulletins an era of advancement will have been posted, that all connected with our institutions should be proud.

We sincerely trust, however, that matters relating to the stewards and purely material interests will be kept out, and above all, the medico-political marks: if they crop in good-bye to the value of anything else that might be there that's good.

C. H. B.

## Periscope.

### PHYSIOLOGICAL.

**Researches on the Connections Existing Between the Nuclei of the Motor Nerves of the Eye on One Side and the Posterior Longitudinal Fascicle and Reticular Formation on the Other Side.**—Mahaim (*Bulletin de l'Académie Royale de Médecine de Belgique*, 1895, No. 5).

The investigations were made by Gudden's method. Two rabbits aged one day, were experimented upon. In the first experiment the author extirpated the right III and IV nerve. In the second animal he extirpated the same nerves and the VI nerve besides. The animals were left alive for four months. Specimens hardened with the bi-chromates, sections stained with ammonia-carmin.

The following condition was found in both cases :

In examining the series of transverse sections from cephalad to caudad one observes a rapid increase in volume of the posterior longitudinal fascicle of the normal side, while that of the operated side receives hardly any new fibres. At the level of the posterior (caudal) border of the IV nerve the fascicle of the operated side is only half the size of that of the normal side. The difference is due to the disappearance on the operated side of the lateral, and to a great extent also of the ventral part of the fibres. On the normal side study of the course of the fibres, which are absent on the operated side, shows that they assume a latero-dorsal direction and dissipate themselves in the reticular formation which they probably help to form. Part of them probably connect with the gelatinous substance of the fifth nerve of the same side. This finds confirmation by the fact that this substance is less developed on the operated side reaching up to a less higher level than that of the normal one.

These researches, the author concludes, prove again the heterogeneous composition of the posterior longitudinal fascicle. At the levels between the nuclei of the 3rd and those of the 5th nerves, the lateral part of this fascicle is composed of fibres which apparently serve to establish a connection between the nuclei of the motor nerves of the eye on one side and the reticular formation and substant. gelatinosi of the fifth nerve on the other.

ONUF.

**Thalamic Heat Centres.**—In the 11th and 12th Band, *Pflüger's Archive*, Prof. S. Taugl has a paper upon heat centres. He made experiments upon horses with an instrument three millimetres in diameter, with which he punctured the brain down to its base through a trephined opening in the skull. After the puncture the temperature was noted in the rectum. Antiseptic precautions were taken to keep the wound free from infection. After death the brain was hardened in alcohol and sections made to determine the site of puncture. He noted that when the puncture was in the anterior end of the thalamus, that the use of temperature was four to five degrees Fahrenheit and remained there only a few hours when it descended.

ONUF.

## PATHOLOGICAL.

**Further Researches on the Ulnar Symptom in Lunatics.**—Goebel (*Neurolog. Centralblatt*, 1895, No. 16).

In the *Neurologisches Centralblatt*, 1894, No. 7, Biernacki published a short notice on analgesia of the trunk of the ulnar nerve as a symptom of tabes. Examination is made in the following manner. The patient's forearm is held semi-flexed on the arm, the physician then takes hold of the patient's elbow and presses with the index finger upon the trunk of the ulnar nerve, where the latter passes in a groove over the internal condyles of the humerus.

Under normal conditions this pressure causes a stinging pain which is felt as far down as the tip of the small finger. The degree of pain felt expresses itself in the physiognomy of the patient, a fact which gains importance in the examination of lunatics from whom we frequently can only in this manner gain information of the effect produced.

G., after the examination of a large number of lunatics (stupor, dementia with excitation, melancholia, confusion, alcoholism, epilepsy, progressive paralysis) reaches the conclusion that the ulnar analgesia is a pathognomonic phenomenon, a helpful test for securing the diagnosis of progressive paralysis in men. It is not absolutely characteristic of this disease as it is also met with in other abnormal mental conditions, especially in epilepsy (it was present in 80. of fifteen cases of the latter disease). When, however, there is suspicion of progressive paralysis, the presence of the analgesia seems to speak in favor of the existence of this organic psychosis, while its absence speaks against it.

G. thinks that it would be worth while to study the value of the symptom for the demaskation of simulators and for the distinction of epileptic from hysterio-epileptic attacks. ONUF.

**Cerebral Palsies of Childhood.**—T. Diller, M.D. (*The Medical and Surgical Reporter*, April 13, 1895).

After a resumé of the subject as discussed in the text-books of Dana, Gowers, Henoeh, Strumpell and others, the author details the histories of seven cases of cerebral palsy, whose most prominent clinical features are shown in the table following:

Case.	Age at onset.	Mental condition.	Moral condition.	Paralysis.	Epilepsy.	Time bet. 1st & 2nd convulsions.	Athetosis.
1	8 months.	Bad.	Bad.	L. H.	Yes.		
2	2 years.	Good.	Bad.	L. H.	Yes.	3 years.	Yes.
3	Birth.	Slight.	Good.	R. H.	Petit mal.	4 years.	Yes.
4	8 months.	Dull.	Good.	L. H.	Yes.	No long interval.	No.
5	6 years.	No change	No change	R. H.	Convulsions only at onset.		No.
6	6 years.	Fair.	Good.	R. H.	Yes.		Yes.
7	1½ days.	Fair.	Good.	None.	Yes.	14 months.	No.

All of these cases, except in birth palsies, began with convulsions, immediately followed by hemiplegia. Two began at eight months, one at two years, and two at six years. In only two cases (5 and 7) was either mental or moral deterioration noted. In all cases, excepting case 5, epilepsy is present. In cases 2, 3 and 7, intervals of three and four years, and fourteen months respectively intervened between the initial and the second convulsion. Athetosis is present in three cases, aphasia in one. MEIROWITZ.

### **Pathology and Therapy of Progressive Bulbar Paralysis.**

—Remak (*Berlin. Klin. Wochenschr.*, No. 2, 1895).

Remak reports the case of a woman twenty-one years old, of healthy parentage, in whom there existed amyotrophic lateral sclerosis with involvement of and paralysis in the upper seventh nerve distribution. This in contrast to the widespread opinion, and it may be said the rule, that the upper facial innervation is never affected in progressive amyotrophic bulbar paralysis.

Remak says that his case demonstrates also that the therapeutic efficacy of electricity cannot to be relegated to mental influence or suggestion. As the result of daily galvanization of the swallowing muscles, an anode of 30 ccm. applied immediately beneath the border of the occiput, a cathode 15 ccm. close to the pomum Adami, and with a current of from 3 to 6 m.a., stable application.

The patient had been reduced to a very unnourished condition on account of the inability to swallow. After a few days the beneficial effects of the electrical treatment were to be seen, and it was not long before the patient could eat and swallow with some degree of comfort. This increased capacity to take food was followed by an increase of bodily weight and a general bettering of nutrition, which continued for several months, when the progressiveness of the disease once more assumed the ascendancy.

J. C.

**Talalgie.**—(*Gazette des Hospitaux*). In a work having for its title, "A Contribution to the History of Talalgie," MM. Brousses and Berthier, of the Lyon's Medical School, have attempted to elucidate the pathology of talalgie. They observed a case where an opportunity to make an histological examination was afforded and as a result have formulated the following conclusions: First, the cases observed had for its essential lesion a localized interstitial neuritis of traumatic origin. Second, the predisposing cause of this affection was an anomaly of the nerve distribution. According to these authors the term talalgie belongs in nosological classifications as an appellation simply to designate a group of affections, badly classed, having but certain common symptoms and differing most frequently and profoundly between themselves as regards anatomical lesions, and requiring a therapeutics as varied as their pathology.

FREEMAN.

### **THERAPEUTICAL.**

**The Employment of Sparks of Opened Induction Coils for Diagnostic and Therapeutic Purposes.**—Sternberg (*Neurolog. Centralblatt*, 1895, No. 13).

If one end of a secondary coil of an induction apparatus is connected with the earth by a suitable conductor, a powerful spark is obtained from the other end, which is produced by the opening induction current. These sparks have the same effects as those of a static machine or of a Leyden jar.

To conduct off to the earth, one end of the secondary coil is connected with a piece of telegraph wire; the free end of the latter rubbed bright with emery or sand paper is wound several times around a water or gas pipe, which previously was rubbed or filed bright so as to secure metallic contact; finally the wire is soldered to the pipe with lead.

The other end of the secondary induction coil is connected in the usual manner with a common conducting wire.

If the free end of the conducting wire is now held near the skin of an individual, sparks will be seen jumping over. But if the free conducting wire is connected with a common well-moistened electrode and the latter firmly applied to the hand, no effect is felt even with full approximation of the coils of the sliding apparatus. In this case the human body acts as a condenser and becomes charged with electricity only



at the surface so that no current passes through the nerves. If a person thus connected approaches another one, sparks jump over to the latter.

If the sliding coil forms part of an "electric table" or "tableau," it is not advisable to make the connection with the earth and with the free conducting wire by means of the two poles of the "tableau," as in this case "influence working" weakens the sparks. To avoid this it is best to connect directly with the two poles of the secondary coil. The described effects can be obtained only when good, powerful apparatus are used; the cells must be of sufficient size and freshly filled.

Practically, S. has made the following applications of his method:

1. For diagnostic purposes to test the sense of pain.

The free conducting wire is not connected with an electrode, but its free end is directly approached to the skin district to be tested. The sparks which then fly over to the skin of the patient are painful; an analgesic district can thus be marked off very conveniently. The evil consequences of the customary test for pain with the needle, bleeding and the risk of septic infection, are avoided by this procedure.

2. For therapeutic purposes.

The free conducting wire is connected with a well-moistened electrode which the physician seizes with his left hand. His right (dry) hand, the palm of which is turned towards the patient is then held over and at a distance of from  $\frac{1}{2}$  to 1 mm. from the region to which the electricity is to be applied. With a sufficient strength of current, that is when the two coils of the sliding apparatus are sufficiently approximated, numerous small sparks fly over from the palm of the physician to the skin of the patient. The latter has a pricking sensation; the physician feels nothing in the left hand (the one which holds the electrode), while in his other (the "faradic hand") he feels a very slight, hardly noticeable effect of the current.

S. has employed this method with good success in the treatment of all kinds of painful diseases, especially of headaches of various kinds. The neurasthenic "head pressure" disappears frequently, the frontal and temporal pain of chlorotics nearly always after one or several sittings. In two cases of cerebral tumor—diagnosis confirmed by autopsy—the headache could be relieved for several months by one sitting. S. does not deny that the therapeutic effects are such of suggestion. This, however, does not detract from the usefulness of the method. ONCE.

**The Treatment of Neurasthenia Insomnia.**—Claus (*Hfen. Klin. Rundschau*). Among the hypnotics recommended in recent text-books on nervous diseases for the treatment of the insomnia that so frequently attend cases of neurasthenia, a prominent place is generally assigned to trional. In this class of cases it is necessary to administer a remedy which will produce a normal refreshing sleep, free from unpleasant after-effects which will act promptly, and be easy of administration, and last, but not least, which can be given for a long time without loss of effect or formation of a habit. Trional possesses all these qualifications, which are demanded of an ideal hypnotic in neurasthenia. Dr. Claus has lately reported thirty cases of neurasthenic sleeplessness, in which it was more reliable and safe than other drugs. It must not be forgotten, however, as Claus takes pains to emphasize that neurasthenics are apt to exaggerate their subjective symptoms, and often sleep better than they pretend—and this fact must be taken into consideration in estimating the results obtained from the hypnotic. In the majority of these cases he finds that the patient falls asleep as soon as he is in bed, but awakes at the end of two or three hours, and can obtain no further sleep for that night. Others remain awake for hours, often until morning. The question now arises, how shall trional be employed in the former class of patients? Shall it be administered before the patient retires, or at the time of awaking? On the ground of his experience Claus concludes that the former plan is to be preferred,

as the sleep produced is much more refreshing. In cases where the patient is restless and cannot fall asleep, larger doses are usually indicated. Another point to which the author calls attention is the injurious effect of insomnia upon the various symptoms of insomnia, especially the dyspepsia. He cites a number of cases in which the relief of sleeplessness under the administration of trional was followed by a rapid improvement of the digestive functions, and with Charcot regards the dyspepsia as secondary to and not as a cause of neurasthenia. The favorable effects of trional also extend to other symptoms of the neurasthenic condition so that it deserves to be regarded as something more than a hypnotic in this obstinate class of cases and may lay claim even to curative properties.

**Note on the Production of Amaurosis by the Extract *Filicis Maris*.**—Mosius (*Bulletin de l'Académie Royale de Médecine de Belgique*, 1895, No. 6)

In the treatment of individuals affected with anchylostomes the application of the extract *filicis moris* has been recommended not only in rather large doses (10 grammes), but several days in succession so as not to destroy the parasites only, but also their eggs. Sometimes visual troubles, amblyopia or even amaurosis have been seen to follow this radical treatment. Usually these disturbances pass away. The author reports two cases, however, in which these disturbances remained stationary. In one case after the administration of ten grammes pro die during three days complete blindness set in, and the patient remained blind. Ophthalmoscopically, first a simple anemia of the disk and retina was observed. Pupils dilated ad maximum, without any reaction. In the course of time complete atrophy of the optic nerves was established.

The second patient had taken ten grammes of the extract on the first day, eight grammes on the second, and the same quantity on the fourth day. The night after taking the last dose amaurotic amblyopia developed in somewhat less degree than in the first case. Pupils dilated at maximum; no response; anemia of the disks and retina. Some improvement of vision took place within months, but only to such degree that he could count fingers at a distance of six metres with the right eye, while with the left he could count them only when the hand was kept close by the eye; recognizes colors except green which appears gray to him. Pupils have remained dilated, but the left shows slight response to light.

M. has experimented with the extractum *filicis moris* upon four dogs. On two of them he effected amaurosis which remained stationary. The result of the microscopic researches of the specimens obtained from these two dogs, which will probably give information of the nature of the disease will be published later.

OUNF.

**Electricity in Medicine.** W. J. Morton, M.D. (*N. Y. Med. Jour.*). Electro-therapeutics in a biological sense is the transformation (by the law of conservation of energies) of electric energy into that peculiar to vital cells. Electricity must not be regarded as an entity, but as having a variety of "properties," such as electrolysis, cataphoresis, etc., each of which may be used, singly or combined. Strong galvanic currents depress tissue nutrition, producing structural changes leading to physiological atrophy. Mild galvanic currents stimulate nutrition and produce hypertrophy, and if alternating cause similar effects to mild continuous currents. The galvanic current and the negative pole is especially indicated in chronic inflammation where newly-formed tissue occurs. Batabolic or destructive events in tissue uniformly present the sign of negativity, that is to say, are at their origin electro-positive. The negative pole is indicated to arrest catabolism, the positive to augment it. The positive pole is rarely indicated, and if so at all, upon the basis of an electrotonic effect to produce sedation of neu-

ralgic pain in superficial nerves. Concerning the faradic current, its main uses are to tetanize muscle and cause sedation of pain. The tetanizing current, as now employed to treat paralyzed muscles, is injurious since it enfeebles the muscle and causes atrophic structural changes. To strengthen a paralyzed muscle a slow rhythm of the faradic—about thirty waves to the second—should be used. In some spastic conditions of muscles (due to paralysis of an opposing group) the strong tetanizing current may be used with advantage to overstimulate and thus fatigue the muscle. The faradic current or static electricity is an adjunct of great efficacy. It evokes the usual nerve and muscle reactions, affords a most convenient means of stimulating the continuous peripheral nerves, producing counter-irritating, reflex, and other afferent effects. It has a local perturbatory action (spark), and it causes profound alterations in the metabolism of the individual, increasing the natural waste products, and diminishing the toxic or by-products. For this reason it is especially indicated, in real or general malnutrition. FREEMAN.

## CLINICAL.

*Diffuse Sclerosis of the Brain and Spinal Cord.*—Burk (*Inaugural Dissertation*, Tübingen, 1895).

Male, good heritage, denied syphilis, intemperate. Disease began in twenty-fifth year, gradually, tremor of right hand; in the beginning intermittent, occurred especially when writing. Nine months later it affected the right elbow joint and gradually became more severe. Two years after beginning of illness the patient felt weakness of left leg, dragged foot when walking. Weakness increased, lessened mobility; no pain or perverse sensations. In his thirty-first year, feeling of weakness in right leg, movements slow, never so severely involved as left leg. A year later the head began to totter and shake, tremor in right arm gradually increased, also the left arm. At first the shaking of head was intermittent, soon increased and became continuous. All disappeared during sleep. In his thirty-eighth year, examination showed in addition to what has been given that there was disturbance of speech, syllables jerky, between individual syllables a toning inspiration was made. For the next eight years the patient was so much better that he could go without a stick. For some months, at the end of that time the tremor and weakness was very much worse and he could walk only on crutches. The speech disturbance more severe, and he also noticed that he saw objects laterally to the field of vision double. Later the disturbance of vision vanished.

In his forty-eighth year, aside from the unarticulated speech, immobility of tongue, the continuous tremor movements of the head which ceased only during sleep. The strength of the arms, especially the left, was decreased, likewise, same of lower extremities. Disturbance of co-ordination in standing and walking; the patient shook so that he could not be trusted alone.

This condition continued about the same up to his sixty-third year. He then complained of some difficulty in swallowing.

In his seventieth year, a careful examination showed sensibility intact; also, intelligence and special senses. Speech more marked disturbance, all symptoms little more severe, symptoms worse on left side; knee-jerk present; no ankle clonus. In his last years, often complained of a bad taste; sense of taste normal. Toward the end there were loss of reflexes, difficulty in swallowing, incontinence urine and feces. Death in seventy-sixth year, symptoms of pneumonia and pulmonary oedema, and weakness of heart after influenza.

Post-mortem showed diffuse sclerosis of brain and spinal cord. Old area of softening in the right angular gyrus. Arterio-sclerosis. The spinal cord somewhat small and flattened, great resistance to the knife

0.1 cutting through it. The brain was small, weighed only 1,100 gms. with the membranes. The sulci gaping and the surface of the brain looking granular on account of small sinkings in.

The microscopical examination showed: Brain uncommonly hard, almost leather-like consistence. Gyri small, white and gray substance sharply differentiated, numerous corpora amylacea. No sclerotic areas or foci to be found, and it could not be said with certainty that there was an increase of the neuroglia or connective tissue. Nerve fibres well preserved; white substance especially showed no deviation from normal; ganglion cells normal; ventricles distended; pia thickened and injected.

In the cerebellum the inner cortical layer in an individual focus looked pathological. The spinal cord, a thin, flattened band, leather-like consistency, hard to cut, showed a thickening of the pia and of the septa which radiate in from it; normal preservation of the nerve fibres, and ganglion cells; white and gray substance very much diminished in size, gray more. No actual increase of the neuroglia; increase of corpora amylacea; hydromelia of the central canal in the cervical enlargement and in the environs increase of the neuroglia. J. C.

**Abortive Acromegaly with Macroglossia.**—Chanffard (*Med. Week*, July, 1895). A patient, thirty-two years of age, who has long been in the habit of drinking and smoking in excess, and was formerly affected with lead poisoning. In addition, he suffers from emotional hysteria and irascibility, with left hemianæsthesia. The writer was struck by the large size of the tongue, which led him to examine him carefully for acromegaly, with the result of detecting: (1) marked prognathism; (2) lengthening of the face; (3) cervico-dorsal kyphosis, with compensatory dorso-lumbar lordosis; (4) hypertrophy of the penis; (5) increase in size of the occiput.

Examination of the extremities revealed no pathological modification, the hands being absolutely normal, as well as the feet.

He suffered, however, from continuous headache and ocular disturbances, which had entailed almost complete blindness of the right eye.

These symptoms justify the diagnosis of abortive acromegaly without hypertrophy of the extremities. That there is also hypertrophy of the hypophysis is shown by the headache, and particularly by the visual disturbances. It is known, indeed, that the latter are of very frequent occurrence in acromegaly, a fact which is explained by the compression exerted on the optic chiasma and nerves by the hypertrophied pituitary gland. The compression is evidenced in this case by congestion of the right optic disc, swelling of the veins, stenosis of the artery, and atrophy of the retinal pigment. The patient has, consequently, arrived at what Tamburrini describes as the hypophysal period of acromegaly.

The relationship between acromegaly and giantism is of interest. Already in 1892, Massalongo looked upon acromegaly as a variety of the latter. Tamburrini compared cases of acromegaly with others of ordinary giantism, associated with hypertrophy of the pituitary gland. Lastly, Brissaud and Meige have recently published a monograph on this subject, in which they urge that acromegaly is giantism in an adult, while giantism is acromegaly in an adolescent.

This patient is far from being a giant, as he is only 1.625 metre in height, and weighs but 56.2 kilogrammes. Were it not for the slight prognathism, which he presents, he would be a well-proportioned man. The tongue, however, is of truly gigantic proportions; but macroglossia appears to be a much more frequent and more marked feature in acromegaly than in giantism. Identifying acromegaly with giantism appears to me, therefore, to be going too far. The writer is rather inclined to look upon these two conditions as closely related and frequently associated, but entirely autonomous. J. C.

**On a Form of Neuritis Caused by Vascular Diseases.**

Schlesinger (*Neurolog. Centralblatt*, 1895, No. 13).

A man, 69 years old, who had been perfectly healthy and free from syphilis, was taken with pain and weakness in the left leg, accompanied by intercostal neuralgia of the left side. Duration of these disturbances about nine months. Without any apparent cause, gradual increase of the symptoms with periods of exacerbation, gradual intense hypertrophy of the left ventricle of the heart. Sudden paralysis of the extensors of the right, later of the left upper extremity and of the peroneal group of muscles; still later paresis of the triceps and deltoid muscles; finally paralysis and rapid atrophy of most of the muscles of the extremities with development of contractures. Lessening of the response to the faradic current, reaction of degeneration in several muscles; diplopia; constant intense sensation of cold in the legs; dissociation of the pain sense and of temperature on the back. Paralysis of sensibility in all qualities at the most distal portions of the extremities; nerve trunk show slight, muscles intense tenderness on pressure; violent spontaneous pains in all extremities; temporary disturbances of the vesical and rectal functions; acute decubitus at right olecranon; chronic progressive fatal course; intelligence undisturbed; duration of the disease about one year.

The post-mortem does not give evidence of any microscopical changes, except insignificant atheroma of the large blood-vessels and moderate emphysema of the lungs. The microscopical examination shows very grave degenerative changes of all nerves with far advanced disease of the vasa nervorum. In all nerve trunks examined, proliferation of the endoneural interstitial tissue, partial shrinkage of the degenerated nerve bundles. The vascular changes affect by preference the arteries, but also the veins; they consist in thickening of the walls in all three layers, narrowing of the lumen often of such degree as to lead to obstruction, and in thrombosis of single blood-vessels. Ascending degeneration of the nerves up to the ganglionic cells of the anterior horns (which latter, however, are not involved), and degeneration of the posterior roots with consecutive ascending degeneration in the posterior columns. Enormous degenerative, inflammatory changes of the muscles with grave pathological alterations of their blood-vessels.

ONUF.

**Tobacco Mania Treatment by Hypnotism and Suggestion.**

—In the *Journal de Médecine de Paris*, March 17, 1895, two cases of the excessive use of tobacco are described, as having been totally cured by suggestion.

The first case was that of a young man 26 years of age, who began to smoke cigarettes in his fifteenth year. His complexion was pale and sallow; his appetite was poor and his digestion feeble; stomach cramps appeared in the morning. Although he was desirous of discontinuing the use of tobacco, he found it impossible to do so owing to an enfeebled will. He was hypnotized several times, and the suggestions were made that tobacco is deleterious to health, that the patient would find the tobacco to be bad, and that he would cease to smoke. The treatment was entirely efficacious, the patient ceasing to indulge in the use of tobacco.

The second case reported by Dr. Voisin is more interesting. It was that of a man 45 years old, who had become hypochondriacal and showed impairment of memory and diminution of moral force. There were cardiac disturbances, pains in the region of the first dorsal vertebra, continual sensation of fatigue, general weakness, emaciation, diminution in the force of the urinary stream, taste slightly obtunded, appetite very poor, and frequent cough. For fifteen years the patient had smoked between forty and sixty cigarettes daily.

The hypnotic state was induced, and at the second seance it was

suggested to the subject that he dislike tobacco, and smoke no more than three cigarettes daily. In two more sittings, it was further suggested that the patient smoke not at all and absolutely detest tobacco. The treatment in this case was also entirely satisfactory, the patient losing all desire for tobacco.

MEIROWITZ.

**A Case of Kahlbaum's So-called Katatonia.**—L. W. Dodson, M.D., (*Med. Record*, July 6, 1895), reports the case of a man aged twenty-seven, who began to suffer from depression and delusions of persecution. After a time he grew worse, refused to talk, laughed insanely, destroyed furniture and would only eat when food was placed in his room at night. Five months later he was confined in bed with the following symptoms: limbs rigid, rhythmical movements of face muscles, causing continual grimacing; muscles of extremities in a state of tonic contraction with marked resistance to passive motion (symptom of negativism). When an attempt was made to move the arm or leg on one side, the corresponding limb on the opposite side moved in unison with it. When the limbs were released, they immediately sprang back to their original position. Cutaneous sensibility diminished. Tendon reflexes absent. At times would suddenly arch the back and support the body on the occiput and heels, remaining in the position of opisthotonos for fifteen minutes. Is usually silent, but at night frequently shrieks, and at times repeats the same profane words over and over. When not watched will gormandize food. All the symptoms are intensified when he is under observation. Under treatment his condition improved somewhat, but was followed by relapse. In this case the alleged causes were isolation and masturbation. The author states that etiologically and pathologically, it is impossible to draw a sharp line between the so-called katatonias and stuporous melancholia with cataleptoid symptoms. Clinically, however, the difference would be obvious to the most cautious observer, and all honor is due Kahlbaum and Kiernan for having clearly and concisely marked out the characteristic features of katatonias.

FREEMAN.

**Anorexia Hysterica.**—Kiesel (*Die Therapie der Gegenwart*, March, 1895), reports the case of a girl, eleven years old, whose mother was hysterical and whose father an habitual drinker, died of phthisis at the time of his daughter's birth. The girl worried much for some years because she could not go to the institute where her sisters were. In a year her character had changed markedly. She became very peevish, retiring and excitable and showed a great dislike towards her mother. At this same time she became very religious and conceived the idea that she ate too much. Consequently she began to fast and rapidly to emaciate. On one occasion she attempted to choke her mother for advising her to take more nourishment. On admittance to hospital she was so thin that her bones stood out prominently, the muscles were atrophied, the skin then dry and brown, but its sensibility normal. The urine was very pale, like water, feebly acid, and contained no albumin or sugar. Her weight was 22,200 grm. For the first few days she refused all nourishment and only took a little milk. The temperature varied between 35 and 37.7°. Many unsuccessful attempts were made at hypnotic suggestion. Forcible feeding was then employed, and at certain times she took small morsels herself. During the first two weeks her weight diminished, but after this she gained rapidly, although she had contracted measles and influenza. She gained 13,250 grm., so that she weighed 35,450 grm. and was unrecognizable as her former self. The family history, the absence of organic disease, the psychical disturbances, the absence of pharynx reflexes and the rapid recovery all point according to K. to hysterical anorexia.

FREEMAN.

## Book Reviews.

A TEXT-BOOK ON NERVOUS DISEASES BY AMERICAN AUTHORS. Edited by F. N. Dercum, A.M., M.D., Ph.D., etc., with 341 engravings and 7 colored plates. 1,056 pages. Lea Brothers & Co., Philadelphia, 1895.

During the past nine years American neurologists, and scarcely more than twenty at that, have contributed to Pepper's System of Medicine, to Keating's Cyclopædia of Diseases of Children, to Hare's System of Therapeutics, to Hamilton's System of Legal Medicine, to the American Text-Book of Diseases of Children, not to mention several others about to be published. Much of this work has been securely buried in these books, so little recognition has it received at the hands of current writers. Dr. Dercum was fortunate in securing so large and so able a list of writers, but it is very evident on an examination of the book that a number of them have gone into the work in a half hearted fashion. A few have been satisfied to transfer, almost bodily, chapters from their own text-books to this one and some have written their articles heatedly and without due regard to their previous good records. Such contributions help to emphasize the fact that this matter of publishing cyclopædias and co-operative text-books is being very much overdone.

It is not possible in this JOURNAL to enter into a discussion of the merits of co-operative works. Lack of proportion and frequent repetition are their chief faults. A better acquaintance on the part of the writer with the special subject of which he treats is supposed to be the chief virtue. Dr. Dercum's book may be said to exhibit both the faults and the virtues to a moderate degree. The salient features of the book will be gathered best by a short analysis of the various chapters.

The introduction is written by Weir Mitchell and the editor. It is needless to say that this chapter is well written, and that the student can gain from it most of the facts regarding the general methods of general examination and the various tests that are to be employed. The study of gait and of the deep reflexes is particularly exhaustive; we cannot say quite as much for the manner in which the electrical reactions are explained, and, we think, a special chapter might have been devoted to the subject of electro-diagnosis and electro-therapeutics instead of appending a chapter on neuro electro-therapeutics at the very end of the book. In the study of sensation special reference should have been made to the recent investigations of Goldscheider.

The editor, no doubt, thought that the examination of the eye from the standpoint of the neurologist would be described in a more authoritative manner by an ophthalmologist than by one of his own coterie, but in this he must have been, and the reader will be, sorely disappointed. The contribution of Dr. Oliver on this subject is on the whole one of the most remarkable that we have ever seen in any standard work. We cannot account for it in any other way than by supposing that the writer did not take the time, or else did not think it worth his

while, to present the subject in a careful manner. He was not only careless in reading proof, but in the entire conception of the article. We will not make too much of the evident errors, as when he states that "from paralysis of the external rectus muscle the eye will be turned outward by the unopposed action of the healthy internal rectus muscle," nor of the fact that he speaks of "lagophthalmos or drooping of the lower eye-lid," nor of his very awkward definition of the Argyll-Robertson pupil, which is defined very much better on page 300. But we cannot help quoting a few paragraphs as examples of the very remarkable English exhibited in these few pages. We are told that "ptosis is both congenital and acquired." The two methods of ophthalmoscopy are described as follows: "One is known as the direct method, as the object within the eye is looked directly at, and the other is described as the indirect method, because an aerial image of the object is what is looked at." Or take this: "Having determined the equilibrium of the pupil, as it is termed, whilst the eyes are in a passive state, as it were, the actions of the sphincter of the iris under the various forms of stimulants that are usually applied to it are next to be tried." The article closes with a paragraph which, if it appeared in a Sophomore essay, would be preserved for time immemorial. After speaking of the various tests which the physician should make, the author continues: "Everything that is relevant should be noted, accurate register of passing symptoms should be taken and the uncertainties of cases so constantly repeated as to obviate all past difficulties. If this be done most carefully and painstakingly it will be a source of great happiness and pleasure to realize that all of the findings have reached a plane of certainty that other observers through carelessness and consequent ignorance have sought for in vain."

There is a decided relief in passing from this article of Oliver's to the chapter on neurasthenia written by the editor. Dr. Dercum's discussion of this disease is very thorough, and his conclusions are those of a man of large experience. He makes an admirable distinction between neurasthenia and hypochloridriasis, and dwells at some length upon gastro-intestinal disturbances so often associated with neurasthenia. He states that a more or less decided diminution in the amount of free hydrochloric acid is noted and that the latter may be entirely absent. We have been struck by the fact that in many of these cases in which there is also a passive dilatation of the stomach and of the intestines an excess of acid is present. It is this class of cases that we have found particularly benefitted by tonic hydrotherapeutic procedures and a vigorous massage of the entire abdomen. In discussing the pathology of neurasthenia Dr. Dercum refers to the researches of Hodge on the changes in nerve cells due to function activity, and infers from his researches and from the discoveries of Mosso, the changes that we should expect in these functional diseases. Mosso conceived the idea that "fatigue altered the constitution of the blood, and he afterwards discovered that the blood of fatigued animals, when injected in an animal at rest, produced in the latter the characteristic symptoms of fatigue." That in purely nervous exhaustion similar modifications ensue, seems a reasonable conclusion. We are glad to see that Dr. Dercum has insisted upon the necessity of a differential diagnosis between encephalasthenia and a beginning parietic dementia. This point of differential diagnosis is frequently neglected in discussions of the two diseases. In the treatment of the disease the rest-cure naturally plays the most important part, but it seems to us to be time to modify the plan, and at least to substitute hydrotherapy for electricity. Dr. Dercum very properly relies upon but few drugs in the treatment of neurasthenia and prefers the bromides to strychnia—a preference which we must confess we are at a loss to explain. Few neurologists will agree



to the recommendation in the last paragraph of this chapter advocating the use of the extract of the testicle in neurasthenia. The author states "that there can be no doubt that even when the possibility of suggestion is excluded, the use of this remedy is of benefit."

Dr. Lloyd's description of hysteria is characterized by great detail and by a critical judgment of the merits of conflicting schools. The author does full justice to sensory changes occurring in hysteria, and very properly attaches the greatest importance to them in proving the diagnosis of true hysteria. Of *astasia-abasia* he has made a purely hysterical condition, and he combats the view of Löwenfeld, that hysteria and hypochondriasis resemble each other in essential points. A few illustrations exhibiting the characteristics of the convulsive stages of hysteria would have enhanced the value of this excellent chapter. Lloyd's remarks on treatment are thoroughly sound. He is of the opinion that "sedative and depressing drugs ought never to be used in hysteria. They cannot control the symptoms except temporarily, and they may even aggravate them permanently. The whole list of bromides, chloral, opiates and antipyrine is to be condemned." The effect of metallo-therapy he attributes entirely to suggestion. He is also opposed to "the barbarism founded upon false pathology which permits the excision of the ovaries for the cure of hysteria."

In writing upon the nervous affections following railway and allied injuries, Dr. Knapp treats of a subject with which his name has been frequently associated. He does not accept Oppenheim's classification of traumatic neurosis as a distinct clinical entity, but maintains that we may have a condition of neurasthenia or hysteria, and even a sclerosis of the central nervous system consequent upon traumatic injury. The repetition, unavoidable in co-operative text-books, is brought vividly to one's mind by the fact that Knapp in his discussion of traumatic hysteria and neurasthenia, has repeated many of the facts which were embodied in the preceding articles by Dercum and Lloyd.

The editor imposed a rather severe task upon Dr. Fisher in asking him to write the chapter on Diathetic and Toxic Affections of the Nervous System. He has been compelled to discuss under an etiological heading diseases which are quite distinct from one another. Thus we find in this chapter an account of rheumatism, gout, uræmia, alcoholism, morphinism, chloralism, cocaineism, lead poisoning, arsenical poisoning and chronic mercurial poisoning. The conception of diathesis as a concrete condition is surely a very curious one. "The relation of diathesis to nervous affections is of great importance in its bearing on a class of diseases which are often not the direct or immediate effect of the poison on the economy, but rather the accompanying or consequent injury to the nervous system." In the short statement regarding diagnosis, Dr. Fisher makes a confession of the difficulties of his task by saying, "that the diagnosis of rheumatic affections is not difficult where the previous history of rheumatism is present, otherwise the symptoms are not diagnostic." The discussion of gout is a little more fortunate, but the writer will hardly wish to be held responsible for the statement "that sudden hemiplegia or paraplegia do not infrequently occur in the gouty as the result of an acute overwhelming of the functional activity of the cord by the toxic agent." Here again he says that "there is little that is diagnostic except the history of the presence of a gouty diathesis, and that this disappears on the exhibition of the appropriate drugs." The pages on uræmia, although but few are more satisfactory. In speaking of alcoholic neuritis, the writer does not state the facts carefully enough when he claims that the lower extremities are first involved and later the upper. The reverse is very often the case. As regards lead poisoning, Dr. Fisher is evidently of the opinion that the poison may attack both the peripheral nerves and the spinal cord, and in this we are inclined to agree thoroughly with him.

His discussion of this special affection may be commended. Of the next chapter on Diseases—the Direct or Indirect Result of Infection, including Cerebro-Spinal Meningitis, Tetanus, Hydrophobia, Tetany and Diphtheritic Paralysis, we may say that it is written with Osler's characteristic terseness, but the chapter does not call for any special analysis since the sections are built upon the same plan as are the chapters in the author's Practice of Medicine. The discussion is thoroughly up to date, however, and in the treatment of tetanus Osler feels warranted in recommending the tetanus antitoxine. In the discussion of tetany, Osler calls attention to "Hoffman's symptom," referring to the increase of the mechanical and electrical excitability of the *sensory* nerves.

Dr. Sinkler has written so frequently and so ably upon chorea that we naturally expected from him the exhaustive treatment of the subject which he has given. The article is an able one from every point of view, and does full justice to the many opposing theories with regard to the etiology and pathology of the disease. We are glad to see that he puts a quietus upon the tendency prevalent in some quarters to attribute this and other neuroses to ocular defects. He states, "that evidence seems lacking that the refraction error is the basal cause of chorea." Sinkler states distinctly that the mental state in the mild type of chorea is seldom changed—a fact that should be emphasized in view of the extraordinary statements to be found in many text-books. In the reviewer's opinion, mental deterioration accompanies only a few of the most pronounced and chronic cases of chorea. The statement of the various pathological findings in cases of chorea is thoroughly unbiassed. Sinkler is not yet ready to accept the theory of microbic infection, and while some of the cases may possibly be explained in this way, it is altogether too early to generalize from a few cases. We do not think that Dana's case can be utilized to prove the microbic origin of ordinary chorea. The reviewer is in entire accord with the author in making rest and quiet the most important features of treatment in cases of chorea. Sinkler is not in favor of large doses of bromides, and while he advocates the use of arsenic, calls attention to the dangers of multiple neuritis following upon the use of extreme doses of this drug. In self-limited diseases, almost any drug may be claimed as a specific, and it is quite safe to say that fully ninety five per cent. of the cases of chorea can be cured by rest, by the simple blood tonics and by nutritious food. The remarks upon hereditary chorea and senile chorea give all the essential details of these two forms. The term electrical chorea is restricted to Dubini's disease, and it is to be hoped that other authors will follow his example in order to do away with the confusion that has arisen from the application of this term to a number of different conditions. The discussion of choreiform affections ends with a description of athetosis. The author concedes that there is a primary athetosis, but the majority of cases are associated with hemiplegia or a diplegia, and the illustrations given in this chapter are of patients afflicted with a unilateral or bilateral spastic palsy.

Dr. Burr has a useful chapter on Local Spasms and Occupation Spasms. In the treatment of torticollis the author favors operative procedures. He believes in stretching or cutting the spinal accessory nerve.

The next three chapters on Functional Tremors, Paralysis Agitans and Epilepsy are written by Dr. Gray. We are compelled to admit that they show altogether too clearly that the author has fallen back upon the chapters in his own text-book, and has not made the discussion of the subject as exhaustive as we would expect them to be in a large work like Dr. Dercum's. This is particularly true of the chapter on Paralysis Agitans. We must take exception to the statement that "in paralysis agitans the prognosis is good under proper treatment." The

author's experience is somewhat unusual, for he states that, "I have seen cases of fifty years duration, in which no impairment whatever could be detected in the mind." Cases of paralysis agitans beginning at the age of twenty, or even earlier, are surely exceedingly rare. The section on the pathology of paralysis agitans is very useful. The author evidently inclines to the theory of senility and gives a good account of the heterogenous findings in the various post mortem examinations that have been made. Dr. Gray recognizes the use of hyoseyamine as well as nourishing and stimulative treatment; among the latter he includes galvanism. The pages on Epilepsy are very familiar indeed, since they are taken with but very few changes from Gray's text-book. We feel that the distinction between focal and general epilepsy has not been insisted upon sufficiently, that in the treatment of the disease special attention should be paid to those cases of organic epilepsy that are not due to traumatic injuries, but are associated with cortical or meningeal disease in early life.

We have nothing but praise for the admirable article of Dr. Brill on Arrested Development, Malformations and Hydrocephalus. We know of no other chapter in any English text-book in which the facts embodied in this chapter are put together in as logical a fashion and in which the chief conditions are as clearly described as here. Beginning with the large defects the author passes to a discussion of Porencephaly, microcephalus and cranial abnormalities, to departures from the normal development of the fissures, to absence of entire parts of the brain, and includes almost every known abnormality of development. Hydrocephalus internus should have had a somewhat more exhaustive discussion, for after all, the condition is associated quite as frequently with acute disease as it is with conditions of arrested development.

In the next chapter by the editor, on General Diseases of the Brain, there is a satisfactory discussion of pachymeningitis, of leptomeningitis of various kinds, meningeal hemorrhage, diseases of the sinuses of the dura mater, cerebral anemia, and hyperemia, cerebral inflammation and brain abscess. A special chapter might have been made of the diseases of the sinuses and of the dura mater, in view of the importance which these diseases have reached since the publications of McEwen, Koerner and others. Like all other recent authors, Dr. Percum has had to include a consideration of cerebral anemia and hyperemia. He states that a diagnosis of these conditions is rarely recorded. We are surprised to find no reference to Geigel's recent studies of cerebral circulation, for they tend to disprove many of the antiquated notions regarding the fluctuating blood supply of the brain and spinal cord.

Dr. Mill's chapter on Cerebral Localization proves how thoroughly he is acquainted with the literature of the subject, and exhibits to a remarkable degree his ability to present the views of adversaries. He states his own theories regarding motor and sensory representation in the cortex, but gives due weight also to those who differ from him. It is a pity that Dr. Mills was not asked to write upon the Anatomy and Physiology of the entire brain and spinal cord, for in that way a very distinct gap might have been satisfactorily filled.

Dr. Dana's chapter on Apoplexy is well written and well illustrated. The treatment of the subject is exhaustive and thoroughly up to date. In discussing the pathology of apoplexy he gives due weight to the various forms of arterial disease, and claims changes in the vessel walls to be the chief cause of hemorrhage. The statement that it is impossible to rupture a healthy artery by any physiological or pathological disturbance, may have to be modified in view of recent publications by Stein and others, tending to prove that sudden increased pressure is after all the most important factor. The truth will probably lie midway between these conditions, and that in the majority of cases both increased pressure and arterial disease are to be held responsible for the

occurrence of cerebral hemorrhage. Dana favors the practice of compression of the carotids upon the affected side in the first stage of cerebral hemorrhage. It is to be feared that this would be a rather dangerous practice in the hands of general practitioners. It seems doubtful, too, whether much more harm than good would not be done by this procedure in any case. With the recommendation that bleeding should be resorted to in plethoric patients with a strong heart action, it is easier to agree, but the author justly enough calls attention to the fact that it is well to be careful with this procedure unless one can be absolutely certain that the case is one of hemorrhage and not of thrombosis.

The discussion of hemorrhage is followed by a succinct account of acute softening of the brain due to embolism and thrombosis.

Dr. Starr has given a careful account of Tumors of the Brain, and includes in his chapter a discussion on cortical localization, in which he repeats some of the facts stated by Mills. This is a matter, however, for which Starr is not to be held responsible. The article is written with the author's usual clearness of diction, and the symptomatology is stated with all needed detail. From the analysis of cases he shows that but seven per cent. of tumors of the brain are open to operation. This makes the prognosis sad indeed, if we consider that of the cases operated upon up to the present time, not more than three per cent. were, or could have been, successfully removed. The author believes that within the next ten years these statistics will be materially changed. We are inclined to doubt this prospect, but even granting it, we must not forget that according to his own statistics over ninety-three per cent. of all cases of brain tumor must prove fatal.

Dercum's discussion of the Cerebral Palsies of Childhood gives a concise but sufficient account of this interesting group of diseases. The article is well written and amply illustrated. The serial illustrations on page 514 are somewhat dramatic in character, but we do not think that they give the characteristic positions of spastic diplegia with choreiform and athetoid movements as well as a few selected photographs would. Dercum does not incline to the polioencephalitis theory of Strümpell, and is in accord with those writers who claim that the acute cases are more apt to be due to vascular lesions such as occur in the adult. His remarks on treatment are good, and we are glad to see that he does not reject the idea of possible benefit to be derived from operative treatment, at least as regards the treatment of epilepsy so frequently associated with these palsies.

The Diseases of the Spinal Cord are introduced by Lloyd's chapter on Malformations of the Spinal Cord, embracing a good account of spinal bifida; he discusses also pachymeningitis and leptomeningitis, and refers to traumatic injuries and caries of the spine and concludes with a few remarks upon hyperemia and anemia. Lloyd believes that it would be best to abolish hyperemia and anemia as designations of special forms of disease of the spinal cord.

This is followed by Morton Prince's discussion of Myelitis. The chapter embraces a careful account of the ordinary symptoms and pathology of the various forms of myelitis. The table on page 566 gives in a very succinct fashion the differential symptoms between lumbar, dorsal and cervical forms of myelitis. The author discusses quite fully the etiology of acute myelitis, and seems to be very nearly in accord with those who maintained that if we set aside the cases due to traumatic injury and those due to syphilis, very few forms remain that could properly be called acute myelitis. Chronic myelitis is also considered at some length, but it is probable that this chapter will in future works be relatively shorter, for many of the forms of disease now included under this term will probably prove to be forms of simple or combined sclerosis or of spinal syphilis. Prince includes a discussion of

Caisson Disease, of Landry's Paralysis, which he is inclined to regard as a toxic affection, involving the motor elements chiefly, and therefore not the spinal cord alone. The chapter contains a good account of syringomyelia and of Morvan's disease, which Prince considers identical with syringomyelia; and he closes with an account of tumors of the spinal envelopes and of the cord itself. The description of the symptoms due to tumor is good, and the chief points of differential diagnosis between tumor and the various forms of myelitis are carefully indicated. With regard to treatment, he suggests specific treatment in the case of syphilitic tumors, and surgical interference whenever there is a possibility of removing an extradural growth. It is well in this instance to insist upon the fact that in spite of the few successful cases that have been reported, the large majority of spinal tumors can not be removed successfully by the surgeon.

It is one of the surprises of the book that these pages of Lloyd and Prince should not have been preceded, instead of followed by the chapter of Peterson, who, by way of introduction to his chapter, gives the fundamental facts of the Anatomy and Physiology of the Spinal Cord. This introduction is well written, but we are a little disappointed in the rest of the chapter, for Peterson evidently must have considered himself limited to a very small space since he was willing to devote scarcely more than four pages to poliomyelitis, only one page to that important subject, lateral sclerosis, only thirteen pages to the consideration of locomotor ataxia, of which a very considerable part is taken up by illustrations. In a book like this poliomyelitis and locomotor ataxia should have received the fullest possible discussion, so that the student could have gone to it with the conviction that he could find an accurate discussion on any one of the many mooted points regarding these diseases. The statement that there are no recorded autopsies in primary lateral sclerosis needs modification in view of the publications of Strumpell. The discussion of the pathology of tabes although altogether too short, is satisfactory since it indicates the more recent theories regarding the changes at the inception of the disease. This chapter should also have included a reference to the hereditary spinal diseases, which might have been considered with Friedreich's ataxia. It is unfortunate that an author who is capable of good work, should have given such brief sketches of the diseases instead of bringing his learning and acquaintance with the literature of the subject to bear upon his discussion of it.

The chapter on Parietic Dementia, written by Dr. Dercum, is one of the best to be found in any English text-book; it is exhaustive and critical, but it leaves the reader with a feeling of regret that the subject of tabes which is so closely related to parietic dementia, was not discussed as liberally. Three times as much space is devoted to parietic dementia as to tabes. This is, indeed, reversing the usual order of things in a text-book on nervous diseases. Dr. Dercum has taken equal pains to make the article on Syphilis of the Nervous System a credit to him, and he has managed to embrace in it the conclusions of most of the recent writers on the subject. We are pleased to note the statement that "the diagnosis of a given case of nervous syphilis should more frequently read multiple cerebro-spinal syphilis, than either syphilis of the brain, or syphilis of the cord." Dercum thinks Erb's type of spinal syphilis worthy of special consideration, but is careful to state that all cases of spinal syphilis do not conform to it. The treatment of syphilis of the nervous system is also given with considerable care. The author is of the opinion that the effects of a thorough mercurial treatment are more persistent than if iodides are used alone, and the reviewer can subscribe to the opinion that the "hypodermic method of administering mercurials has nothing special to recommend it in the

treatment of nervous syphilis, the most rapid impression being made by the method of inunctions."

Very few text-books contain a more thorough account of the diseases of the peripheral nerves than this work of Dercum's. A few charts giving the anatomical distribution of the various plexuses would have contributed to a still clearer understanding of the subject, but it is a curious fact that even the largest text-books on nervous diseases, containing innumerable illustrations of the anatomy of the brain and spinal cord, give but little attention to the anatomy of the peripheral nerves.

Dr. Sinkler has written well on Neuritis, Multiple Neuritis, Tumors and Mechanical Injuries of the Nerves.

To Dr. Schweinitz's discussion of Diseases of the Optic, Oculo-Motor, Pathetic and Abducens Nerves we have referred above. The discussion is an excellent one in every sense and a most useful one. In speaking of the etiology of primary atrophy of the optic nerve, the author has shown his appreciation of the needs both of the student and the specialist. The article is amply illustrated and will tend to enhance the author's reputation. Diseases of the cranial nerves not included in Dr. De Schweinitz's article are discussed in a special section by Dr. Herter. The discussion is adequate in every case and embraces the most recent views regarding the diagnosis and treatment of these affections. Dr. Herter also has his fling at the eye-cutting neurologists and ophthalmologists. In speaking of the cutting operations upon the extrinsic muscles of the eye for the relief of trigeminal neuralgia, he suggests that these have been extensively performed without the slightest justification and often with distinct injury to the patient. In view of the unanimous opinion of all respectable authors on this subject, we are surprised at the audacity of those who still recommend this procedure for the relief of all sorts of possible and impossible conditions. In a special chapter Dr. Herter has gone quite extensively into an account of the diseases of the cervical nerves. It is this chapter that would have been greatly benefited by the introduction of anatomical charts.

Sub-acute progressive polymyositis, and progressive muscular dystrophies, progressive neurotic atrophy and Thomsen's disease, as well as arthritic muscular atrophy, are discussed in one chapter by Dr. G. W. Jacoby. Although rather condensed, the articles embrace all the essential points. Jacoby believes that the changes in the peripheral nerves are undoubtedly the primary and more important ones in progressive neurotic atrophy. This belief has been strengthened by the publications of Dubreuilh, Hoffman and others. The latest contributions of Marie and Marinesco again throw some doubt upon this point, for in the cases examined by them the chief changes were in the spinal cord. It is probable that in this form as in so many other affections, one or several parts of the neuron may be involved in this disease.

Dr. Jos. Collins's article on Trophic Neuroses is well written and well illustrated. The account of the rare disorders included in this chapter is altogether sufficient, and will help to make some of these diseases more intelligible than they have been hitherto. The description of Raynaud's disease, fully illustrated as it is, is particularly good. Adiposis dolorosa, as first described by Dercum, is accorded the distinction of special description, and the chapter closes with an account of scleroderma, morphea, facial hemiatrophy, facial hemi-hypertrophy and local hypertrophies.

Dr. Burr has written a special chapter on the Trophic Diseases associated with pathological changes in the thyroid gland. It embraces a short account of myxoedema, of cretinism, and exophthalmic goitre. As regards the pathology of the last named disease the author concludes that "evidence is quite strong that the cause of the disease is a poison

originating in the thyroid and acting mainly upon certain structures in the medulla."

Headache, migraine, vertigo, insomnia and other disorders of sleep are discussed by Dr. James C. Wilson. This chapter should have been introduced in the earlier part of the book in which general functional diseases are discussed; it is, on the whole, satisfactorily written. It must again be a disappointment to the "eye-cutting" specialists that Dr. Wilson has little or nothing to say regarding the beneficial effect of these operations in the treatment of migraine. Wilson believes that chloral is the surest of modern hypnotics. He claims that chloralamid is less active but altogether safe in proper doses. Sulfonal is said to be uncertain in its effects but "the virtues of euophen, chloral hydrate, somnal and tetronal as sleep compelling drugs are now established;" but some of us probably consider sulfonal, on the whole, more satisfactory than several of those included in this list.

There are few men who can speak with more authority on the surgery of the brain and spinal cord and nerves than Dr. Keen of Philadelphia, and his article bears the stamp of a large personal experience and an intelligent interest in every aspect of the subject. Keen gives a special consideration of Lannelongue's operation for microcephalus; he stands quite alone in his recommendation of this procedure. He claims that a moderate number of children show some improvement and that in a few it has been very great. In the discussion of hydrocephalus he includes Quincke's method of puncture in the lumbar region. The discussion of the indications and the technique of operations in cases of cerebral abscess and of thrombosis of the lateral sinus is timely and sufficient. There commendation that in removal of tumors of the brain the operation be done in two stages is one well worthy of consideration. The first stage is to consist of opening the skull and dura and determining the site, character and size of the tumor, and then temporarily closing the wound; if operable the removal of the tumor may be undertaken in from three to six days later by re-opening the wound. This would seem to us to apply only to cases of large tumor; if the tumor be of small size the little additional shock at the time of operation will not interfere seriously with the prospects of recovery. The surgeon and the neurologist are indebted to Dr. Keen for his succinct account of Hartley's operation and for his remarks on surgery of the spinal cord and of the peripheral nerves. Keen refers incidentally to an operation by Abbe and one by himself in cases of syringomyelia; he concludes, fortunately, that it would seem best not to operate in such cases. We have never been able to understand the ground on which such an operation is attempted if a diagnosis of syringomyelia has been made.

The book closes with Dr. Jacoby's chapter on Neuro-Electro-Therapeutics, of which it is sufficient to say that it includes most of the essential points on this subject.

This extensive review of the articles in Dercum's Text-Book was warranted by the long list of well known names associated with that of the Editor. It is proper to acknowledge before closing that much excellent work has been condensed in these pages, but the reviewer believes that the majority of the contributors to this volume will agree with him in the statement that American Neurology is more apt to be advanced by the publication of a comprehensive text-book by a single author than by further encyclopædic or co-operative works. For the present Gowers' Manual supplies every need, but we should be pleased to see it supplanted in the course of the next five years by an American work which shall prove as reliable as the volumes of Gowers have been.

R. Sachs

INJURIES AND DISEASES OF THE NERVES. By John K. Mitchell, M.D. Lea Bros. & Co., Philadelphia.

Our understanding of the retrogressive and regenerative processes following the injuries of nerves, has been so much enhanced by the recent experimental investigations of Howell and Huber, Vanlair, Nissl, and others, that clinical application of the knowledge gained will excite our liveliest interest. Mitchell's book will, therefore, certainly be welcomed by the profession. In certain points the clinician is, of course, at a disadvantage over the one who gains his results by means of experiments. The latter can shape the primary conditions more or less according to his will and study the pathological changes resulting in all cases and at any stage. The clinician must accept conditions as they are and has not frequently the opportunity of controlling the correctness of his conclusions by autopsies and microscopical examination of specimens. This disadvantage makes itself somewhat felt in M.'s book as pathologic-anatomical data are given in only two cases. Otherwise the book is a valuable addition to neurological literature, giving a large number of interesting clinical experiences and useful suggestions.

The text is divided into seven chapters preceded by an introduction.

The first three chapters treat on the various lesions of nerves, viz., contusions, commotions, complete and incomplete sections; some pages are devoted to injuries of the spinal cord and its environs.

Each chapter begins with general remarks and conclusions drawn from the cases which latter are reported at the end of the chapter. The reports of all the cases which the author had opportunity to observe himself are very exact, giving all details to make the histories complete. Those which lack in exactness were obtained from other sources, many data had even to be gathered from written reports of the patients.

The description of each case begins with a statement of the diagnosis and a concise summary of the prominent symptoms. At the end of each history we find remarks intended to point out the peculiarities and characteristics of the case described. This arrangement is a great convenience for the reader, who can easily get a general orientation on the case before entering upon details which are of special value to him.

Many interesting facts are learned from these histories, for instance, the peculiar thermal effects observed in some cases, where at the time when sensation began to return temperatures around 110° F. caused scalding; then the various forms of misreference of sensation seen in other cases, etc.

To devote a special chapter to the description of the sensory tests used and to a general discussion of the value of such tests in general might, perhaps, not have been amiss. On the other hand; we must greatly approve of the author's warning not to rely too much upon sensory tests in judging the return of function in an injured nerve, but to give more importance to the electrical reactions as a criterion. M. gives interesting and valid proofs of the fallacy of sensory tests in this regard.

The fourth chapter treats on ascending and migratory neuritis. The views displayed here will probably find opposition from many sides. Neuritis is evidently considered as identical with nerve inflammation. This absolute identification is objectionable in the light of the recent researches which have shown in how many cases of neuritis nutritive or degenerative changes, not inflammation, were the essential feature. Proliferation of the surrounding neuroglia and connective tissue—findings upon which the author lays much stress as manifestations of an inflammatory process—so frequently follows the degeneration of nerve



fibres or cells that its occurrence cannot be accepted as an absolute proof of inflammation.

It is admitted by M. that ascending or migratory inflammatory neuritis very seldom occurs as a sequel to nerve injuries. It has been observed to take place when there was an open nerve wound. In such cases an invasion of microbes along the lymphatic paths of the nerve evidently was the cause. But it is difficult to conceive, how a pale scar of the hand involving a small branch of the ulnar nerve should have been in existence for twenty years without producing any symptoms and should then, after the lapse of this time, call forth an ascending nerve inflammation, involving all nerves of the arm. A careful analysis of the facts in this case (No. 39) will demonstrate that the degenerative changes found in the nerves of the arm which finally had to be amputated, might very well have been the descending effects of the nerve stretchings and nerve resections performed previously. At least it is astonishing that the author emphasizes "the nearly perfect correspondence between the pathological observations and the clinical symptoms," without alluding to the possibility that at least part of the pathological changes might have been the secondary effect of the operations mentioned. Whether the case in question was one of neuritis must remain a matter of conjecture; the foregone operations complicated the conditions in such a manner that histologically the presence of neuritis could not be proven.

Case 3, cited as one of neuritis, may also excite doubt; at least another diagnosis was made here by Dr. Jacobi. Case 40 was too complicated to represent a pure type.

The symptom-complex of neuritis was evidently present in some cases, but no convincing proofs of the occurrence of ascending or migratory nerve inflammation are given.

The chapter on miscellaneous cases gives interesting histories of injuries which offered unusual peculiar features.

In the sixth chapter degeneration and regeneration of severed nerves are discussed. The clinical side is thoroughly described with communication of many valuable data.

The seventh chapter informs us as the plan to be pursued in the treatment of nerve injuries. The surgical treatment is given due consideration, the bad prognosis which joint stiffenings and the lesions caused by long continued rest give, is called attention to. ONCE.

## THINKING, FEELING, DOING. By E. W. Scripture, Ph.D., (Leipzig.) Flood & Vincent, Meadville, Pa.

The author who is well known as the director of the Psychological Laboratory at Yale, presents us through the Chautauqua Century Press, a curious text-book upon what he calls the *new* or experimental psychology.

The first statement that attracts our attention is that his is the first book written in the English language upon this subject. This seems strange, as another work familiar to us and to at least thirteen thousand others, should have been to him also, viz.: "Practical Lessons in Psychology," by W. O. Krohn, Ph.D., (Yale.)

Dr. Scripture addresses his work to the people. Dr. Krohn more particularly to the school teacher, and claims to avoid "pedagogical consciousness," "icy cognitions of thought," "primordial elements," and tries to have his book characterized by rather a "practical ring" than a scholastic rattle."

Scripture follows this lead also, to write in every day English and

as he puts it very cleverly: "have not tried to clothe my ignorance in the 'multitudinous syllabifications, and frango-maxillary combinations that pass as philosophic English.'"

These two works are on similar lines. Prof. Krohn's should have the credit of being a pioneer in the new psychological text-book making. Scripture's style is very curious, he opens with a sort of "now you see it and now you don't" way, and while trying to be instructive immediately prepares every reader to enter a realm of prestidigitation, magic and occultism. The first paragraph reads, "Eyes and No Eyes journeyed together. No Eyes saw only what thrust itself upon him; Eyes was on watch for everything." Answer to this conundrum: "Eyes used the fundamental method of all knowledge—observation or watching." Repeatedly he opens up the subject in this manner, and then with a hoppity-skip, go-as-you please or any other way, draws a hasty inference, or explains a misty or complicated fundamental law. Definitions are enigmatical to our author he prefers figures of speech. Let us for a moment draw attention to page 89, the very best chapter of his book on the subject "attention," or as he writes it, atTENTion. "In the first place what is attention? It is a very sad fact, but I cannot tell what it is; the innumerable psychologies attempt to define it, but when they have defined it, you are sure to know just as much about it as before." He then follows this up with illustrations and details of what it is, covering about twenty pages, and they are about the best of the book.

His treatment from beginning to end while interesting to those having some knowledge of the subject of experimental psychology can not help but mystify the general reader at times. The endeavor on the author's part to be simple, has ended in making him often rather ridiculous and ambiguous. He has written for the people in rather a photographic style, and the prints when most necessary to be in detail are blurred and clouded.

Is this the way to culminate a chapter on memory? "Indeed we might say that memory is the ostensible friend who insists upon presenting us with a house bountifully furnished with the skeletons of past sins, but who in old age turns us out into the cold night of forgetfulness when we would gladly remember even the sins." Read this to any one and see if they can form an idea of memory from it. He begins the chapter, "If I were writing a dictionary I would define memory as that portion of mental life about which everybody has been talking for three thousand years without telling us anything more than any body of common sense knows beforehand." He certainly in that case ought to write sense.

The author has given the public at least a good peep into the laboratory of the advanced psychologist and endeavors to interest him in the work. Crude may be the manner, but the fact remains and that fact is, much depends upon the people understanding that the future education of the child depends upon the "child study" and the needs of growing nature and the mental make-up.

Never mind if our author has drawn wholesale upon such predecessors as Wundt, he gives him in the main credit, though not stopping by the way to note the quotations. We may say all this and even the style of the presentation may be criticised, yet if the book is read carefully every one can find much not only to interest and instruct, but they will be convinced that the book has a place and will do good. The arrangement, printing, marginal references, and illustrations are of first-class order, and side by side of its already successful competitor Dr. Krohn's work, it should be found upon the book-shelf of every teacher in the world.

BROWN.

PRACTICAL DIETETICS, WITH SPECIAL REFERENCE TO DIET IN DISEASE. By W. Gilman Thompson, M.D., Professor of Materia Medica, Therapeutics and Clinical Medicine in the University of the City of New York. 8vo. 801 pages. Illustrated. D. Appleton & Co., New York, 1895.

The author of this work is known as a talented physiologist and clinician, whose writings have always been scientific, original and attractive. The special object of the book is to teach how the sick should be fed, for in the author's opinion this subject has been greatly neglected in medical treatises, by hospitals and in the training of nurses.

Dr. Thompson does not acknowledge any special diet system as a cure-all, or the specific influence of any one food in the treatment of disease; but he believes in a rational method based upon clinical experience, with a sound knowledge of pathology and the physiology of digestion.

The first part of the book is devoted to foods, and food preparations, including their elementary composition, force-producing power, economic value, classification, etc. The author favors a reasonable allowance of meat in health and believes that a mixed diet is the best, yet he considers that most people eat too much meat and not enough vegetables. Although it is usually thought that nitrogenous food is concerned in tissue formation and hydrocarbons in force production, yet he believes that some energy may also be derived from nitrogenous material.

The article on milk is very complete and fully up to date. An account of the Strauss' Milk Depots is given, and also an interesting description of the modern milk laboratories, with illustrations. Adults who are able to eat any kind of food should, according to Dr. Thompson, abstain from raw milk, as it is liable to produce that form of indigestion known as "biliousness," and besides favors constipation.

A list of the numerous food preparations on the market is given, and their advantages, disadvantages, and supposed composition are set forth in a perfectly impartial manner.

Part two treats of stimulants, beverages and condiments. Concerning stimulants the author says: "It the almost universal experience that food and drink merely to satisfy the craving of physical needs does not wholly satisfy the desire for occasional invigoration, for restoration after fatigue, for support during sustained muscular exertion, for an incentive to activity, and for conviviality. In some form or other, although in greatly varying degree, a stimulant is demanded by almost every one to meet the emergencies with which he is, from time to time, confronted." He believes alcohol is wholly unnecessary in health, but that its life-long use in moderation, may not necessarily injure some persons, while in others it produces disease. In a number of diseases he considers it of absolute service and a necessity to prolong life. Its action as a food may, he thinks, be exerted through modification of oxidation processes, or by preventing the burning up of other food materials, which are thereby enabled to be added to the bodily elements. He finds reason to believe in addition, that alcohol may also be directly stored in the body in the form of some other product.

Part three is devoted to the subject of cooking, food preparation, preservation, and the quantity required to maintain the body in vigor.

Part four consists of a section on the foods required for special conditions.

Part five contains in a condensed but well-written form, the essen-

tials of the physiology of digestion, and the conditions which especially affect the digestive functions.

Part six treats of the general relation of food to special diseases, and diseases which are caused by dietetic errors.

Part seven is concerned with the administration of food for the sick. It contains much that is valuable, especially to the nurse, who, as Dr. Thompson says, has a far better opportunity than the physician to judge of the patient's appetite and study his whims and fancies in regard to food.

Part eight is devoted to diet in disease. This is the most important portion of the book and two hundred and ninety pages are given to the subject. In those diseases where several high authorities hold opposing opinions, as in gout, diabetes, etc., these various theories are impartially stated. Much useful matter is presented here regarding the important rôle played by diet in diseased states, and the subject is reviewed in a very comprehensive manner. A full description of the various diet systems and special diet is also included.

Part nine contains numerous rations and dietaries of representative hospitals and government institutions, besides an excellent article on the feeding of infants and young children, and the various dietetic cures. The subject of diet for athletic training is also mentioned, and the dietaries of the Yale boat crew and football teams are given. To this is added an appendix containing numerous useful recipes for invalid foods and beverages suitable for fevers and convalescence from acute illness.

The whole work is certainly a very valuable and highly practical one and fulfills well the requirements for which it was intended. There is a full index, and the printing and binding are admirable.

A. FREEMAN.

LECTURE ON APPENDICITIS AND NOTES ON OTHER SUBJECTS. By Robert T. Morris, A.M., M.D., Fellow of the New York Academy of Medicine, American Association of Obstetricians and Gynecologists, American Medical Association; Member of the New York State and County Medical Societies, Society of Alumni of Bellevue Hospital, Linnean Society of Natural History, etc. With illustrations by Harry Macdonald, M.D. New York. G. P. Putnam's Sons, 1895.

The author of this book of 159 pages has had exceptional opportunities for studying the various subjects of which he treats, and his ideas are set forth in his usual clear and concise manner. As he states in his preface, "this collection of lectures includes the substance of my teaching on the subject of appendicitis at the Post-Graduate Medical School of New York, and . . . the substance of many of the notes has appeared in various periodicals." This fact, however, does not detract in the least from their value, and the book will be read with interest by surgeons even though they may not fully agree with the author in many of the ideas set forth in the notes.

The illustrations, of which there are many, are the work of Dr. Henry Macdonald, and are finely executed from specimens and dissertations furnished by the author.

The first chapter deals with the preparation of the surgeon and patient for operations in general. The author keeps pace with the most advanced ideas on asepsis, and in addition strongly advocates certain theories of his own.

The anatomy and pathology of the appendix vermiformis cæci, together with its contents, are ably treated in the second chapter; and the author makes the interesting statement that the nearest approach to a "seed" he has been able to find in an appendix, "was a piece of apple-core encrusted with phosphates."

Chapter III. is an admirable dissertation on appendicitis; which the author defines as "an infective, exudative inflammation of the appendix vermiformis cæci." After describing its cause, and the manner of entrance of the infection, the pathology of the disease is given in plain terms, the text being rendered more interesting by the photo-micrographs and other illustrations. Three very good points which are forcibly brought out are, first; the little reliance which can be placed upon the temperature in making the diagnosis; second, the marked disproportion which frequently exists between the pulse and the temperature; and third, the fact that the presence or absence of an inguinal tumor is not important. He makes a comparison of the death-rate in cases treated medically and surgically, and then details a typical case under each method of treatment, the comparison being strongly in favor of early surgical interference.

In the fourth chapter the author gives a short history of the advance in popular favor made by the operative treatment of appendicitis in the last five years. His description of the operative technique leaves little to be desired. He strongly recommends the short incision; the proper length, to his mind, being one inch and a-half. He says that this gives all the room necessary for cases, even with dense and extensive adhesions, and the shortness of the incision, together with accurate and careful returning of the incised structures, favors the production of an "evanescent scar," and greatly shortens the period of the patient's confinement to bed. "An inch-and-a-half in a week-and-a-half" being not at all uncommon in his cases.

An interesting report of the author's first hundred consecutive cases of operations for appendicitis then follows, which shows four deaths in the first twenty-five cases; two deaths in the second twenty-five, and only one death in each of the third and fourth sections; while there were no deaths among the fifty-nine simple cases (*i. e.*, those without general infection.)

Chapter V. consists of notes on some twenty-eight different subjects, notable among which are: "Another Method for Palpitation of the Kidney," "The Drainage Wick," "Is Evolution Trying to Do Away With the Clitoris?" and "Ovarian Transplantation."

December, 1895.

GAZZAM.

MYXEDEMA AND THE THYROID GLAND. By John D. Gimlette, M.R.C.S. (Eng.); L.R.C.P. (Lond.) J. and A. Churchill, London, 1895.

The little volume before us had its origin in an augural thesis written in Portuguese, in accordance with the rules of the medical school of Lisbon. It was afterwards modified and published in English in the hope that the general practitioner might find it a convenient epitome of the present state of our knowledge on the subject. This object on the part of the writer has been fully attained. The subject is considered in three parts; first, "Topics Relative to Myxœdema," in which the history, etiology, symptomatology, complications and varieties, diagnosis, and clinical cases are fully considered. Secondly, "Topics Relative to the Thyroid Gland," in which its anatomy, histology and physiology are considered. Part III. deals exclusively with the pathology and treatment of myxœdema itself.

We are glad to note that while full credit is given to Dr. Ord, that the part played by Sir William Gull in first calling attention to this disease, is fully recognized. "To him," as the writer states, "belongs the honor of the authorship of the first pathological treatise, and to the Clinical Society the satisfaction of having stimulated the first clinical studies of this disease, which have been crowned with such remarkable results."

The clinical description is full and satisfactory and this is true of the subjects treated in the book throughout. An excellent index is also added.

F. X. DERCUM.

# APPENDIX TO DUNGLISON'S MEDICAL DICTIONARY. Twenty-first edition. Philadelphia. Lea Brothers & Co., 1895.

This latest addition to the original Dunglison's Dictionary consists of twenty-four large octavo pages, and brings the work abreast of the times.

It contains a large number of new words and names, notably chemical and bacteriological, as would be naturally inferred considering the rapid strides these two sciences are daily making, and is a valuable addition to this most excellent work.

GAZZAM.

# THE ANNIVERSARY OF THE STANDARD DICTIONARY.

The Funk & Wagnall's Company celebrated on November 27, the first anniversary of the completion of their "Standard Dictionary," by putting to press the 90th thousand of this great work. This is a very large number of dictionaries to print in a single year. The publishers' mathematician has figured out that, if these 90,000 sets were piled flat, one upon another, they would reach nearly seven miles in height; and the printed pages, if laid end to end, would extend over 40,000 miles, one and three-fifths times around the globe!

But the most significant of the triumphs of the first year of this remarkable dictionary, and the most gratifying to Americans, is the wonderful reception given to the work by the most exacting of the linguistic critics in England. Especially is this so when we remember how reluctant, naturally enough, the English are to look to a foreign country for a dictionary of their own tongue. It is something extraordinary for an American work of this kind to elicit words of such enthusiastic praise as those uttered by such scholars of the Oxford University as Professor Sayce and Max Muller, and well-known scholars of other English universities, and from such journalistic critics as those of the *London Standard*, *Saturday Review*, *Notes and Queries*, *Nature*, *London Times*, *Westminster Review*, *Athenæum*, *Mark Lane Express*, *Scotsman*, *Liverpool Post*, *St. James Budget*. The latter closes his critical review with the following superlative indorsement:

"To say that it is perfect in form and scope is not extravagance of praise, and to say that it is the most valuable dictionary of the English language is but to repeat the obvious. The Standard Dictionary should be the pride of literary America, as it is the admiration of literary England."

May the triumphs of the second year of this dictionary equal those of the first; they could not well exceed them!

## Miscellany.

### A CORRECTION.

Dr. Leo Stieglitz's article in the November number is only an abstract of his paper presented to the American Neurological Association ; by some means this fact was not stated.

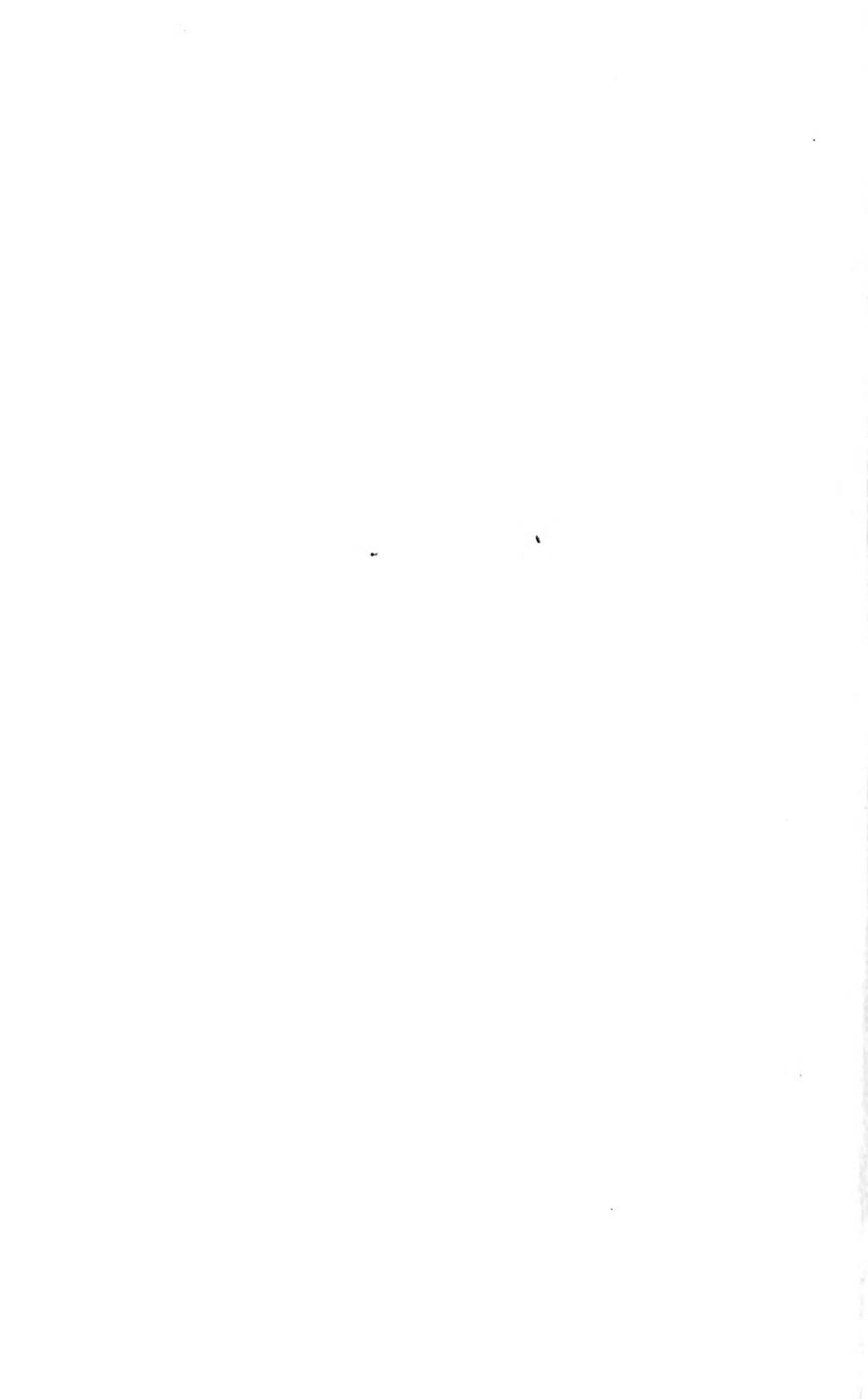
### ANNUAL ANNOUNCEMENT.

In bidding good-bye to the old year, and the close of our twenty-second volume, a sense of pride compels us to draw attention to our increasing importance and the evident fact, as shown in our recorded pages, that America can boast of its neurological work. In our adieus we must not forget to thank Dr. Joseph Collins for his most practical aid, but also to Dr. B. Onuf, a no mean modicum of praise is to be bestowed, for his most valuable contributions. Their work has been of value and in many ways, our readers will feel who have watched their years task, that this recognition is due them, at least. While assuring our friends that we intend to add to our coming year's volume many original papers of importance, we will also assure them that our periscopic work will be of unusual importance and greater usefulness, and we take a great pride in announcing the acceptance of the charge of this department by Dr. A. Weiner, 113 W. 77th street, of this city. There is a need for the "newer" pathology and psychology, and we intend to devote more space for their consideration in 1896. There will be an extension of the department pertaining to work among the insane by the presentation of various administration, clinical, pathological and psychological studies and of notable cases, under the able direction of Dr. R. M. Phelps, of the Rochester State Hospital for the Insane and an efficient corps selected from many representative asylums. It can be assured that our readers will feel the earnest spirit and receive a true picture of the advancement and standing of the

best alienistic work in America, beside, in "abstract form, the best work of our friends abroad. This work will be more fully outlined in the January number. The sciences of biology and comparative neurology in the near future will have proportionate attention and comfortable quarters with their sister sciences. It is the desire of this JOURNAL to crystalize into compact form all that goes to make up the necessities of the worker who desires to keep abreast of neurological sciences of the day. It requires much work and expense and we do not consider it but just to those who do this labor to ask practical assistance in all ways and at all times. It is difficult under any circumstances for a truly scientific periodical to exist and it is not unkind to any other class of journals to plead for prominence. In the selecting of your journals for the coming year place us first upon the list and obtain for us the subscription of every friend you can influence. This seems to some to "smack of the shop" instead of philanthropic science, but the work horse needs provender and that of the right quality.—[ED.]







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